VOLUME 12 NUMBER 9

SEPTEMBER, 1922

# Phytopathology

Official Organ of the

## American Phytopathological Society

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PUBLISHED MONTHLY BY THE SOCIETY

OFFICE OF PUBLICATION

3 West King Street, Lancaster, Pa.

Entered as second-class matter at the post office at Lancaster, Pa.

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PHYOPATHOLOGY

VOLUME XII NUMBER 9

SEPTEMBER, 1922

# STEM AND FRUIT BLIGHT OF PEPPERS CAUSED BY PHYTOPHTHORA CAPSICI SP. NOV.[1]

LEON H. LEONIAN

WITH TWO FIGURES IN THE TEXT

In the fall of 1918 the writer found that a disease was causing considerable damage to the Chile pepper plants on the farm of the New Mexico Experiment Station. A preliminary examination revealed nothing more than an Alternaria, and although this fungus was isolated readily, repeated inoculation experiments with it gave only negative results. It was concluded, therefore, that this organism was merely a secondary invader that followed the injury caused by the primary infecting agent. A year later the same disease reappeared in the field and assumed quite serious proportions. Other fields in the neighborhood were found to be generally affected with it. This time isolations were made from very young lesions. After the surface of the infected tissues was carefully washed with a mercuric chloride solution, flamed forceps were inserted beneath the epidermis, just on the border of diseased and healthy regions, and a small bit of tissues was removed and planted in a poured plate of cornmeal agar. The fungus which eventually grew out of the diseased material was found to be a species of Phytophthora, and when pepper plants were inoculated with it 100 per cent infection was the invariable result.

## THE DISEASE

This disease appears on the branches as a blight, and on the pods as dry lesions of various sizes. If not followed by an attack of Alternaria the market value of the diseased pods is not impaired. When the Alternaria attack is not severe, only an experienced observer can detect any external indication of the disintegrated tissues beneath the epidermis and signs of blackish mouse-gray mycelum of the Alternaria that stuffs the inside of the pod.

[1] The writer is indebted to Dr. C. H. Kauffman for the critical reading of the manuscript of this paper.

The disease generally appears anytime after May when the warm and rainy season begins, and continues until late fall. The first indication of it on the pod is a small, water-soaked, dull green spot which in mild cases does not enlarge very much. In more severe cases elongated lesions which eventually may involve one fourth or more of the pod develop. Very rarely does a natural infection extend throughout the fruit, and in no case has the writer observed a lesion which encircled the pod like a band. The advance of the infection is much more rapid along the stem-end and blossom-tip of the pod than it is along its circumference. A natural infection by this organism is not indefinitely progressive, but for some unexplained reason it eventually comes to a sudden halt; consequently the lesions are narrow, long, and sometimes extend the entire length of the pod. The infected tissues soon dry, bleach to a dull straw color, sink below the level of healthy tissues, and become parchment-like (Fig. 1). The seeds also become infected; then they turn brown, shrivel, and in severe cases die; or they may retain their vitality if the infection is confined to the seed coat only.

FIG. 1. Tip of a Pepper Fruit Showing a Typical Lesion.

Often the fungus passes from the fruit to the branches; then it grows rapidly and involves the younger and tender parts. However, it often extends to the larger branches which are completely girdled and finally killed. Usually after the fungus reaches the older parts or the main stem, its growth stops, so that frequently only one branch, or one half, or even one third of the plant is killed, while the remaining part stays healthy. More frequently the branches become infected not through the fruit but directly at their base by means of the zoospores of the pathogen. A ring of blighted tissues then appears, and the parts above

it soon wilt. If the primary infection is on the main stem, the entire plant is killed; but if it starts on a branch, it moves downward, and either before or after it reaches the main stem, its growth ceases. It is an interesting phenomenon that the infection on the branches progresses around rather than up and down in narrow lesions as is true in the case of infection of the fruit.

## THE FUNGUS

The fungus responsible for the disease of Chile peppers is a species of Phytophthora, and according to the classification of Rosenbaum (3) it falls in the Phaseoli group because of its basal antheridium. A morphological comparison with the other members of the group shows that this fungus is apparently a new species for which the name of *Phytophthora capsici* is proposed. The gnarled mycelium of this fungus is very distinctive. Of all the eleven species of Phytophthora studied by Rosenbaum (3), *P. syringae* is the only one which produced a "tuberculate" growth on potato hard agar. *P. syringae*, however, in addition to its small zoosporangia, forms the antheridia on the side of the oogonia, whereas *P. capsici* has very much larger zoosporangia, and basal antheridia. *Phytophthora parasitica*, *P. phaseoli*, and *P. infestans* of the *Phaseoli* group can also be eliminated because of their smooth hyphae when grown on potato hard agar, the much smaller size of their zoosporangia, and because of the lack of ready oospore formation in case of *P. infestans*. Of the remaining two organisms *P. erythroseptica* has a broad and flat papilla in contrast to the raised and very prominent papilla of the zoosporangia of *P. capsici*. The last member of the Phaseoli group as given by Rosenbaum, *P. arecae*, forms a characteristic vesicle into which the zoospores escape. No such vesicle is seen in *P. capsici* upon the germination of its zoosporangia. Two apparently new species of the Phaseoli group have been reported recently. One of these affects the rhubarb, and the other causes a rot of the tomato fruit. But since these organisms still remain undescribed, no comparison is possible at present.

**P. capsici** sp. nov.

Sporangiophores branched; sporangia generally ovoid, varying in culture to elongated ellipsoid, subsphaeroid, irregularly elongate with intermediate forms; papilla very prominent, either single and apical, or sometimes up to three and variously disposed; germination normally by zoospores, under special conditions by germ tubes; size of sporangia extremely variable, 35–85 μ or even 105 x 21–56 μ, averaging 60 x 36 μ; oospores formed on submerged mycelium, abundant on oatmeal and corn-

meal agars, slightly wrinkled, brown, semi-transparent, 25–35 μ; antheridia basal: mycelium often gnarled, becoming densely tuberous under certain cultural conditions; the tuberous outgrowths sphaerical or ovoid, minute, 5–8 μ, of the same shape as sporangia, richer in protoplasm and darker in color than the mycelium, often very numerous, giving rise to large, grape-like clusters, germinating by germ tubes; no chlamydospores observed. Parasitic on stems and fruit of *Capsicum annuum*. State College, N. Mex.

The characteristic tuberous outgrowths (Fig. 2, c) appear on a great many media, potato hard agar included, but they attain their maximum growth in a solution[1] which has been developed and used by the writer in connection with some other investigations. Only very few sporangia are formed in this solution, but when the mycelium is transferred from this medium to either distilled water or to some nutrient solution which induces sporangia formation, these tuberous bodies germinate by a germ tube which elongates into a sporangiophore, and gives rise to a sporangium (Fig. 2, d). When transferred to distilled water, the protoplasm of the mycelium, as well as that of a number of these outgrowths, migrates into the sporangiophores and becomes separated from the empty hyphae by one or more cross walls. This, however, is not the case when the hyphae are transferred to a favorable nutrient solution; here a normal growth precedes the sporangia formation. These tuberous outgrowths must be considered as a distinct morphological characteristic of this species because of their regular occurrence on a wide variety of nutrient media. Similar bodies have been observed by Ward (5) in *Phythium gracile*. Butler (1) found "toruloid structures" in Pythium. Rosenbaum (3) reported the "gnarled and tuberculate" appearance of the mycelium of *Phythophthora syringae* when this fungus was grown on potato agar, and he used this characteristic to separate *P. syringae* from all of the other species which he studied.

## INOCULATION EXPERIMENTS

Inoculation work with this fungus has covered a period of three years. Field work was done during the summer of 1920 by the writer, and by Mr. W. A. Archer during the summer of 1921. Greenhouse inoculations have also been made by the writer throughout 1921 and 1922 at Ann Arbor, Michigan. The results have been uniformly positive. The fungus is capable of attacking the host through sound tissues. Under the climatic conditions of New Mexico the first visible lesions on the

[1] $KH_2PO_4$ 1. 2 grams, $MgSO_4$ 0.6 gram, peptone 0.6 gram, maltose 6.25 grams, malt extract 6.25 grams, distilled water 1000 cc.

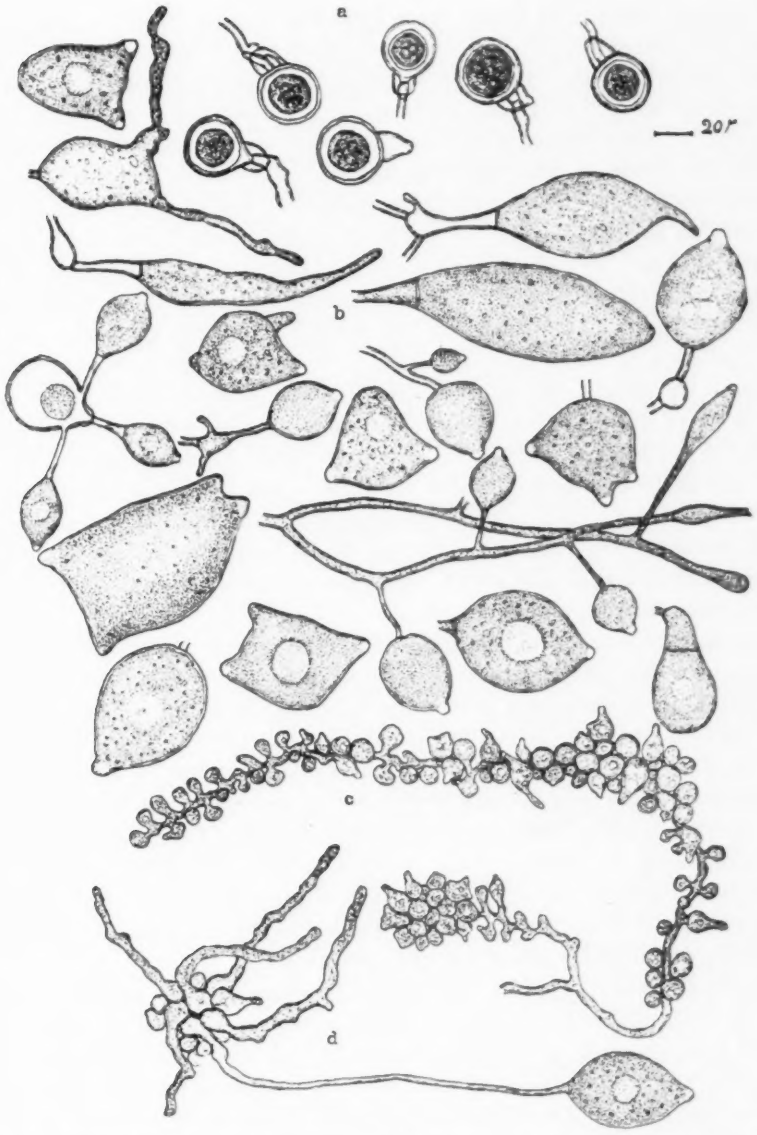


FIG. 2. Phytophthora capsici sp. nov. (a) Oospores. (b) Sporanzia; Different Types and Stages of Developement. (c) Tuberous Outgrowths Formed on the Mycelium. (d) The Tuberous Outgrowths Germinating; One of Them has given rise to a Sporangium.

fruit appear within 24 hours after inoculation and within a period of three days most of the pod becomes infected. Shortly thereafter no sound tissue remains on the pod which is at first soft and pulpy, but gradually dries and shrivels. Meanwhile, if the drying does not proceed too rapidly, the sporangial masses are borne on the surface of the pods within the folds of the wrinkled tissues. Just why infection should progress so rapidly in the artificially inoculated fruits, and why it should advance so slowly and restrict itself to limited areas in case of natural infection, is not very clear, unless it be assumed that mass action plays an important rôle. Bits of mycelium containing a number of sporangia have been used as inoculum in the artificial inoculation experiments, while in case of natural infections it is most likely that only a single zoospore constituted the original inoculum. Smith (4) states that mass action plays an important rôle in the infection brought about by some bacterial pathogens. Heald (2) observed that in artificial smutting at least 0.5 gram of powdered smut well distributed to each 100 grams of seeds is necessary to produce maximum smutting, and that with small spore loads certain varieties of spring wheat remained smut-free. This indicates that either a multiple infection or mass action by the spores is the cause of this phenomenon.

The first indication of the disease on the woody stems can be seen within a week from the time of inoculation. At first a brownish discoloration appears which gradually enlarges, and in another week a dark brown band of tissues girdles the stem. On the tips of younger branches, however, the infection manifests itself within 24 hours. Then the fungus moves upward and downward, passes from branch to branch, and finally ceases to make further progress in spite of the most favorable environmental conditions. In case of greatly under-nourished and weakened plants in the field, and of pot-bound, sickly looking plants in the greenhouse there is no arrest of the disease, the entire plant, roots and all, being invaded by the pathogen and killed.

Under ordinary field and greenhouse conditions the pathogen does not cause a root rot. Repeated inoculations of the soil in which the plants were growing, gave negative results. When diseased plants were buried in the soil in direct contact with the roots of healthy plants, no losses occurred. When after a time these plants were dug out and examined, only a few of the roots were found to be diseased, and it was noticed that the infection, after having made some headway, had stopped, just as in the case of the branches. In another experiment some of the roots of a healthy plant were exposed, inoculated with the mycelium of the pathogen, and again buried in the soil. Later when these were

examined, the disease was found to have progressed from the tips of the finer roots to the larger ones, and then to have come to a sudden halt. This strange phenomenon apparently is not due to the woody nature of the tissues alone, nor is it caused by the death or by the loss of the physiological efficiency of the pathogen, but apparently is due to a local immunity presented by the host. It was noticed that on the branches, after the infection had stopped its progress, the live tissues which immediately adjoined the killed ones had assumed a dark purplish color. In one experiment when this dark region was used as a court of infection, even when it was wounded and stuffed with the mycelium of the pathogen and kept in a saturated atmosphere, no infection occurred. Three attempts at different intervals gave similar results. When, however, the tissues just below this dark region were inoculated, infection resulted and in due time the plant was killed. The immunity, the toxic agent, or whatever term we might choose to apply to this inhibiting factor which is developed only by vigorously growing plants, is highly localized and does not diffuse in the tissues. It is also probable that structural changes, caused by the action of the fungus upon the plant, are responsible for this local immunity.

### DISSEMINATION

The seeds located just beneath the diseased lesions of the pods are seen in various stages of infection. Some are brown and badly shriveled; others may or may not show very distinct external signs of the disease, but when they are planted in nutrient agar they give rise to the mycelium of the pathogen. Some of these seeds when planted in agar germinated in spite of the fungus which was growing out from the seed coat. The young sprouts, however, were soon destroyed by the hyphae. Thus it becomes apparent that infected seeds constitute important agents of dissemination for the fungus. Diseased pods, as a rule, are not discarded by the growers because, as stated before, their diseased condition is hardly noticeable externally. Since the infection invariably extends to the seeds, it follows that if the diseased material finds its way into hot beds, cold frames, or the field, the pathogen immediately starts growing. It may attack some of the roots, without, however, killing the young plants, or it may be carried to the field with the infected soil which clings to the roots. Thus new centers of infection form. Once established in the soil the pathogen may become very difficult to eradicate. It is a matter of common observation that the pods situated on the lower branches are the ones to become diseased, and that the initial infection, at least in the majority of cases, starts near the blossom end.

Since the tip of the fruit usually hangs down, the blossom-end is in the path of the zoospores which are forced up by spattering rain drops. Similarly stem blight usually start at the lower parts of the branches or stems where the zoospores are most likely to find a favorable lodging.

## CONTROL

In view of the foregoing facts the chief control measure should consist in careful seed selection especially if the fungus is not already established in the field. In case the soil is already infected, spraying with fungicides is recommended. While the writer has tried no spraying experiments, it is obvious that zoospores formed in the soil and carried up to the plants are responsible for the infection. A careful spraying schedule, if followed properly, should not fail to give good results.

## SUMMARY

1. A disease which attacks the pods and the branches of Chile peppers in New Mexico was found to be caused by a new species of Phytophthora.

2. The disease manifests itself in the form of dry lesions on the pod. The younger branches are killed outright, while on the older parts of the hosts the blighting of the tissues brings about a rapid girdling.

3. The disease is not indefinitely progressive; the infection stops after reaching the older parts. Artificial inoculations of the sound tissues at the point where the disease stopped, failed to cause infection, while the region a little below this point failed to exhibit any resistance to the pathogen.

4. The fungus can only affect a few of the younger roots.

5. The seeds in the pods become infected and thus serve to disseminate the organism. When infection is confined to the seed coat, the power of germination is not interfered with.

6. Seed selection and spraying with fungicides are recommended as control measures.

CRYPTOGAMIC LABORATORY, UNIVERSITY OF MICHIGAN.

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# THE INFLUENCE OF SOIL TEMPERATURE UPON THE DEVELOPMENT OF FLAX WILT

L. R. Jones and W. B. Tisdale

With One Figure in the Text

In 1914–16, W. H. Tisdale working at the University of Wisconsin (4) made a study of the nature and inheritance of resistance in flax to the wilt disease caused by *Fusarium lini* Bolley. While growing plants in the greenhouse for these experiments he (5) noted marked differences in the rate of wilting of susceptible flax plants growing at different distances from the heating system. Upon investigation, the temperature of the soil nearest the steam pipes was found to be several degrees higher than that more remote from the pipes.

Following out the suggestion that soil temperature might be an important factor influencing the rate of infection, Tisdale devised a simple apparatus for holding the temperature of the soil below that of the room. From trials made with this he reached the conclusion that the critical temperature for the infection of flax by *Fusarium lini* is between 14° and 16° C. The disease became serious with plants in control pots with temperatures at 19° to 21° C. Because of the lack at that time of adequate apparatus for maintaining constant soil temperatures, the minimum temperature for occurrence of the disease was not exactly determined and no effort was made to learn either the optimum or maximum temperature limits for disease development. Following the perfection of the Wisconsin soil temperature tanks (2), the writers during 1921 extended the studies upon the disease through a wider range of soil temperatures, and the (2) data obtained are here presented.

The flax seed used was the susceptible strain, C. I. No. 3, furnished by Mr. J. C. Brinsmade of the Office of Cereal Investigations, U. S. Department of Agriculture. The soil was obtained from a well drained marsh field at Madison which had previously been successfully inoculated with a pure culture of the flax Fusarium. This originally came from the North Dakota material furnished by Professor H. L. Bolley. The water-holding capacity of this soil was 67 per cent (40 per cent based on wet weight) and the moisture content was kept at 25 per cent during the experiments, a condition favorable for the flax plant. A uniform amount of the soil was placed in each culture can (8 x 12 inches), and those to be used as controls were steamed for five hours at one or two

pounds pressure and allowed to stand for a week before planting. After they had been in the tanks for a sufficiently long period for the temperature to become adjusted, one hundred flax seeds were planted in each can. The experimental soil temperatures ranged in a graduated series from 12° to 38° C. with 4° or 3° intervals.

The first trial was conducted in April and it was repeated in May with essentially like results. These are combined in table 1.

TABLE 1

*The percentage of flax seedlings developing wilt at differenpt soil temperatures. Based on two experiments in each of which the plants were grown for 24 days.*

| TEMPERATURE OF SOIL (°C.) | TOTAL NUMBER PLANTS | TOTAL NUMBER PLANTS WILTED | PERCENTAGE OF WILT |
|---|---|---|---|
| 12 | 170 | 0 | 0 |
| 16 | 153 | 48 | 31 |
| 20 | 154 | 104 | 68 |
| 24 | 151 | 147 | 97 |
| 28 | 129 | 123 | 95 |
| 31 | 129 | 103 | 80 |
| 34 | 100 | 67 | 67 |
| 38 | 36 | 0 | 0 |

Since so high a percentage of wilt developed at 16° C. it seemed probable that the critical temperature was distinctly below this. In order to learn this more definitely a third experimental series was run in the same way limited to two soil temperatures, the first 14° to 15° and the second 16° C. The results were that at 16° C., ten plants out of 45 plants, or 22 per cent, wilted within the 24 day period. This was in essential agreement with the results from the first two trials. At 14° to 15° C., however, out of 40 plants only a single one showed slight symptoms of wilt in the same period. The lower critical soil temperature for disease development seems therefore to be about 14° C. The delicacy of the temperature balance was further shown when at the end of the above trial period of 24 days the pots which had been held at 14° to 15° were raised to 16° C. At the end of one week 15 per cent of these plants showed wilt symptoms.

These results are graphed in figure 1. They are in general accord with those of W. H. Tisdale (5) at the lower range of temperatures and add interesting data concerning developments at the higher range. It is noteworthy that in this case as with the other vascular Fusarial diseases tested in the Wisconsin soil temperature tanks (6, 1) the disease is almost as sharply limited in its occurrence at the upper temperature range as at

the lower (see table 1 and figure 1). A comparison of the temperature range of the disease with W. H. Tisdale's results with pure cultures of *Fusarium lini* on agar plates (5) shows that this Fusarium flax wilt disease behaves much as does the Fusarium cabbage yellows disease. Flax wilt developed at the highest temperature (34° C.) at which the parasite grows in pure culture on agar. The optimum temperature for the disease development practically coincides with the optimum for the cultures of the fungus (24° to 28° C.). At the lower temperature the growth of the fungus may occur at a slightly lower temperature (12° to 13° C.) than the disease development (14° to 15° C.).

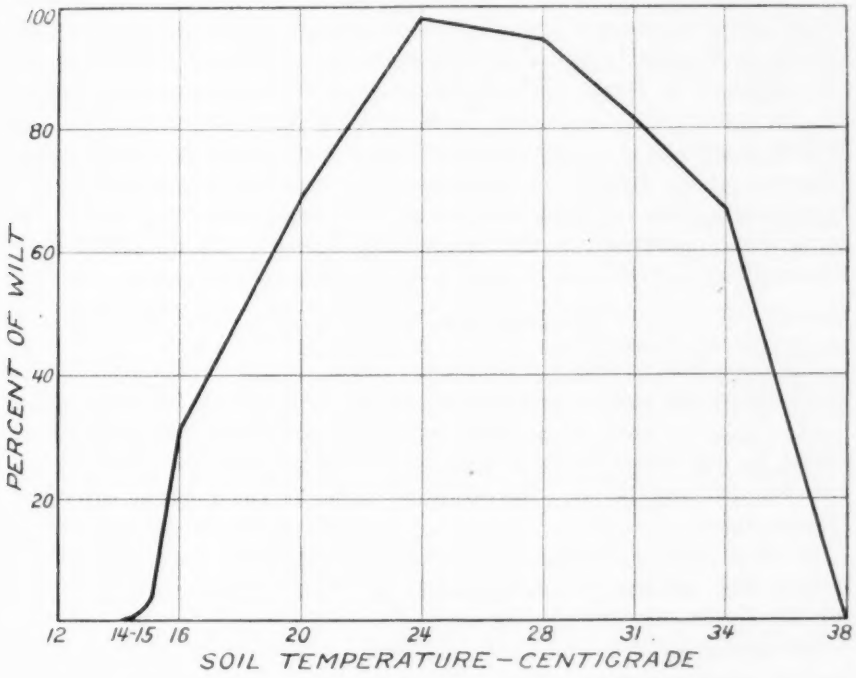


FIG. 1. Graph showing the per cent of wilt developing in flax seedlings at different soil temperatures. For details see table I and the accompanying text. Note that the minimum or "critical temperature" for disease development is about 14 degrees, the maximum apparently 38 degrees or below and the optimum for extreme wilt development between 24 and 28 degrees C.

It is of further interest to note that this is considerably lower than the corresponding lower critical temperature for the occurrence of the tomato wilt disease, caused by *Fusarium lycopersici*, which Clayton (1) has shown to be about 19° C. It is also somewhat lower than that for the cabbage yellows, caused by *Fusarium conglutinans*, which Gilman and W. B. Tisdale define as about 16° to 17° C. The correlation of these

critical temperatures seems to be closer with the temperature relations of the normal host than with those of the corresponding fungi in pure culture on agar. Thus while all these Fusarium species show similar pure culture temperature relations it is evident that the tomato is normally a higher temperature plant than cabbage or flax. It is also noteworthy that these lower temperature ranges as defined by the present experimental method bear a relation to each other which corresponds in a general way to the distribution of these diseases in the northern Mississippi valley. Thus the tomato wilt finds its northern limit of aggressive field development south of the latitude of Chicago; the cabbage yellows, while at its worst in the second tier of states (Indiana, Illinois, Iowa), reaches into southern Wisconsin and Minnesota; the flax wilt is serious to the northern limits of North Dakota. Stoa's (3) discussion of conditions in North Dakota indicate that the average temperature for the month of July at the North Dakota Agricultural College, the hottest month of their summer, practically coincides with this optimum temperature for disease development. Stoa notes with flax wilt, as has been noted with cabbage yellows in Wisconsin, that the disease is particularly destructive during certain seasons when midsummer temperature is high, and is less severe during cool summers.

## SUMMARY AND CONCLUSIONS

(1) Flax wilt caused by *Fusarium lini* is readily influenced in its development by soil temperature as was first shown by W. H. Tisdale.

(2) From his work, using only the lower soil temperatures of limited range, he concluded that the critical low temperature for infection lay between 14° and 16° C. He did not determine the relation to higher temperatures.

(3) The present experiments using the Wisconsin tank method included trial cultures at soil temperatures of approximately 12, 14, 16, 20, 24, 28, 31, 34, and 38 degrees C.

(4) The disease was entirely inhibited at both the lowest and the highest of these temperatures. The minimum or "critical" soil temperature for the disease is about 14°, with 38° C. as the maximum, and the optimum for extreme disease development about 24° to 28° C.

(5) The flax plant will develop in Fusarium infested soil without wilting at both the higher and the lower temperature extremes. Germination is retarded at 12°, but once started the plants make a good stocky growth. At 38°, the growth is weakly. The best normal vegetative development occurs in the neighborhood of 20°, that is, distinctly below the optimum for the wilt.

(6) The temperature curve for the disease corresponds closely with

that for the growth of the parasite. The optimum for disease development, 24° to 28° C., is at the same time the apparent optimum for the vegetative development of the fungus in agar culture.

(7) The field evidence as to the distribution and seasonal variation in severity of the disease is in harmony with these experimental results since the evidence for North Dakota where the disease occurs indicates that flax wilt is aggravated by hot seasons and that their midsummer temperature corresponds closely with the optimum for the wilt.

(8) The data are now available for a comparison of the temperature relations of the very similar Fusarium diseases of the flax, cabbage, and tomato. The behavior of the respective parasites in pure cultures on agar is practically identical. On the other hand the host plants have quite different temperature relations, the tomato being favored by distinctly higher temperatures than either of the others, and, as between the latter, flax seems to be a slightly cooler temperature plant than cabbage. It is possible that there is a natural correlation between this host relation and the fact that the lower or "critical temperatures" for the occurrence of their respective diseases stands in like sequence, that of tomato being about 19°, of cabbage about 16° to 17°, and of flax about 14° C.

(9) The geographical distribution of these diseases in the Mississippi valley also seems to be influenced by these temperature relations. The tomato wilt is a distinctly southern disease with its northern range in the latitude of central Illinois; cabbage yellows is at its worst in the second tier of northern states (Ohio to Iowa) with its northern limit in the latitude of southern Wisconsin; flax wilt reaches to the northern boundary of the United States and probably into Canada.

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# A PHOMOPSIS IN GRAPE FRUIT FROM THE ISLE OF PINES W. I., WITH NOTES ON DIPLODIA NATALENSIS

WM. TITUS HORNE

WITH PLATES XXVI AND XXVII AND ONE FIGURE IN THE TEXT

On October 30, 1917, Mr. Frederick Maskew, in charge of plant quarantine work for the State Commission of Horticulture at San Francisco, sent several grape fruits to the Division of Plant Pathology at Berkeley for advice as to whether certain spots (Pl. XXVI, fig. 4) should be classed as melanose (12, 6, 9, 7, 10). The fruits were from a shipment said to come from the Isle of Pines, W. I., the fruit wrappers bearing the mark "Isle of Pines Grape Fruit, S. A Girard & Company, Sole Distributors."

The fruit was of excellent quality and, aside from the spots in question, had not more than the anticipated blemishes for West Indian grape fruit. These blemishes are treated in a separate paper (11).

The spots were small red brown dots rather evenly scattered, mostly on the blossom end of one fruit. In mature fruits the causal fungus of melanose, *Phomopsis citri* Fawcett, is believed to have died out, and accordingly no attempt was made to study this fruit farther. As far as known *P. citri* had not been reported from the Isle of Pines or Cuba.

On the following day, examination of the suspected grape fruit shipment was made with Mr. Whitney of Mr. Maskew's office. A number of additional specimens were taken including two fruits with a tan-colored rot. These fruits were numbered 6 and 7 and were put in moist chambers in our laboratory for study. Cultures were made from them and kept until the summer of 1918 when they were dried out.

Number 6 was a well developed specimen of Diplodia rot (3, 7, 1, 2) apparently little changed except in color, the clear yellow replaced by tan with some dark shadings and the surface gloss unbroken. Number 7 was in a much earlier stage of rot, only part of the surface being involved, but not certainly distinguishable from 6. Both fruits were kept in moist chambers and various tissue plant cultures made from them. Number 6 gave cultures of a fungus which was evidently *Diplodia natalensis* I. B. Pole-Evans, and readily produced st mend rot (4) in grape fruit and oranges on inoculation in the laboratory (Pl. XXVI, figs. 2, 3 and Pl. XXVII, figs, 7, 8, and 11).

Number 7 gave no culture of Diplodia but a more delicate white fungus, a Phomopsis, (5, 6, 8) which appeared regularly but was frequently overcome by contaminations, especially in media rich in nutrients (Fig. 1, a and b, and Pl. XXVII, figs. 9, 10, 12 and 13). In the later stages shadings of a smoky uniform brown appeared below the surface but not the black subsurface color shown by 6. When in the early stages of deterioration number 7 was cut in halves and left in the moist chamber. White mycelium appeared generally over the cut surfaces but there was no blackening as occurs with Diplodia mycelium.

### CULTURE CHARACTERS OF THE PHOMOPSIS.

On slant tubes of agar with very little nutrients the growth is very delicate, colorless, becoming barely brown tinted, filling the medium with a little white aerial mycelium at the top and a few scattered dark brown sclerotial bodies less than 0.5 mm. in diameter.

On nutrient agar in slant tubes the surface is covered with a fine white matted mycelium. Dark brown bodies occur, and a general brownish somewhat flesh colored pigment spreads into the medium (Pl. XXVII, fig. 9).

On green orange twigs flamed quickly and dropped into sterile water so as to leave about 2 cm. of twig above the liquid, growth was excellent. Cut surfaces become covered with white matted cottony mycelium, the unbroken epidermis becomes brownish black, lightly over-spread with fine appressed mycelium, and broken by numerous small conical pycnidia evenly scattered over the whole surface (Pl. XXVII, fig. 10) As the liquid dries successive interrupted pellicles are formed. They are white to yellowish and smoky brown. There is apparently no growth in the liquid which becomes greenish amber. Some dirty white beads form on the lower pycnidia. Lower pycnidia (newer and moister) have the scolecospores or paraphyses predominating, 20 to 32 by 1 μ, ( (Fig. 1, b). Upper pycnidia have mainly spores 5.6 to 8 by 2.2 to 3 μ (Fig. 1, a).

The fungus was grown on a number of media without difficulty. Slight alkalinity causes it to form a small and weak colony (Pl. XXVII, fig. 12) while slight acidity is not detrimental (Pl. XXVII, fig. 13). Limits of tolerance were not determined.

### INOCULATION EXPERIMENTS WITH PHOMOPSIS.

Inoculations were made on grape fruit and typical stem-end rot developed in several specimens (Pl. XXVI, figs. 5 and 6). The fungus was reisolated and grown in culture in the laboratory at room temperature, giving the characteristic features. About three months elapsed in

all cases between the time of inoculation and the development of undoubted stem-end rot. Fruits were kept in moist chambers in the laboratory and many were lost with contaminations. Successful inoculations were made with both mycelum from agar culture and with spores placed on the calyx and on the wounded calyx scar. None of the inoculations on oranges were completely conclusive. Some were probably successful but were interfered with by contaminations. In the late phase of Phomopsis rot brownish black points emerged through the surface of the fruit and white mycelium spread from these (Pl. XXVI, fig. 6) covering the whole fruit with a finely felted mycelium in which black bodies were prominent (Pl. XXVI, fig. 5). In some cases straw colored beads appeared on these bodies. The fruit very gradually became soft and collapsed leaving the outer shell the last part to disorganize.

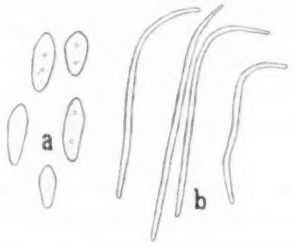


FIG. 1. a. Spores of Phomopsis from Isle of Pines from culture on green orange twig, × 1000. b. Scolecospores (or paraphyses) from same culture as a. × 1000.

INOCULATION EXPERIMENTS WITH DIPLODIA.

Diplodia inoculations were uniformly successful, the stem-end rot well developed in about one month in moist chamber in the laboratory. In later phases of Diplodia rot considerable subsurface black color developed and strong mycelium tufts grew out and later spread over the fruit in an undulating coarse cottony cover which became black except the outermost hyphae which remained gray (Pl. XXVI, fig. 2). Conspicuous sclerotial bodies up to 1 cm. tall and 0.5 cm. broad appeared in some cases (Pl. XXVI, fig. 3).

Most of the culture work reported is based on homogeneous and not single spore cultures and was intended as preliminary work only. While it would be desirable to repeat many of the details of this study it seems better to give out the results than to wait until the opportunity comes to

secure more of this material from the Isle of Pines. The fungus appears sufficiently distinct to be recognized as a new species and I suggest the following specific name with brief description.

**Phomopsis caribaea** sp. nov. Phomopsis isolated from grape fruit affected with stem-end rot, from the Isle of Pines, W. I. Spores from green orange twig in culture 5.6–8 x 2.2–3 μ, scolecospores 20–32 x 1 μ. Growth in nutrient agar culture differs from *Ph. citri* Fawcett in being more vigorous and retaining its filamentous aspect, not becoming finely granular or limelike. Causes stem-end rot of grape fruit in artificial inoculation.

Type specimen, a culture similar to that shown in plate XXVII, figure 10, is deposited in the herbarium of the University of California. Cotype material from the same fruit but a different isolation is being sent to the New York Botanical Garden and to the Bureau of Plant Industry, Washington, D. C.

The generous assistance of Dr. H. S. Fawcett in these studies is gladly acknowledged.

## CONCLUSIONS

A species of Phomopsis was isolated from grape fruit from the Isle of Pines, W. I.

This fungus is capable of producing stem-end rot in grape fruit but under laboratory conditions develops very slowly. It can probably also produce stem-end rot in oranges.

In green orange stems in moist tube culture it develops vigorously.

In nutrient agar culture it appears to differ from *P. citri* Fawcett in being more vigorous and in having the surface of the colony show fine matted filaments instead of a "chalky" surface.

Certain blemishes, tear stains and rots of grape fruit in Cuba and in the Isle of Pines might well be studied with this Phomopsis in mind, as well as deficient moisture or plant food in the soil, exposure, mite and insect injury. The fungus in question is considered to be distinct and the name *Phomopsis caribaea* is suggested for it.

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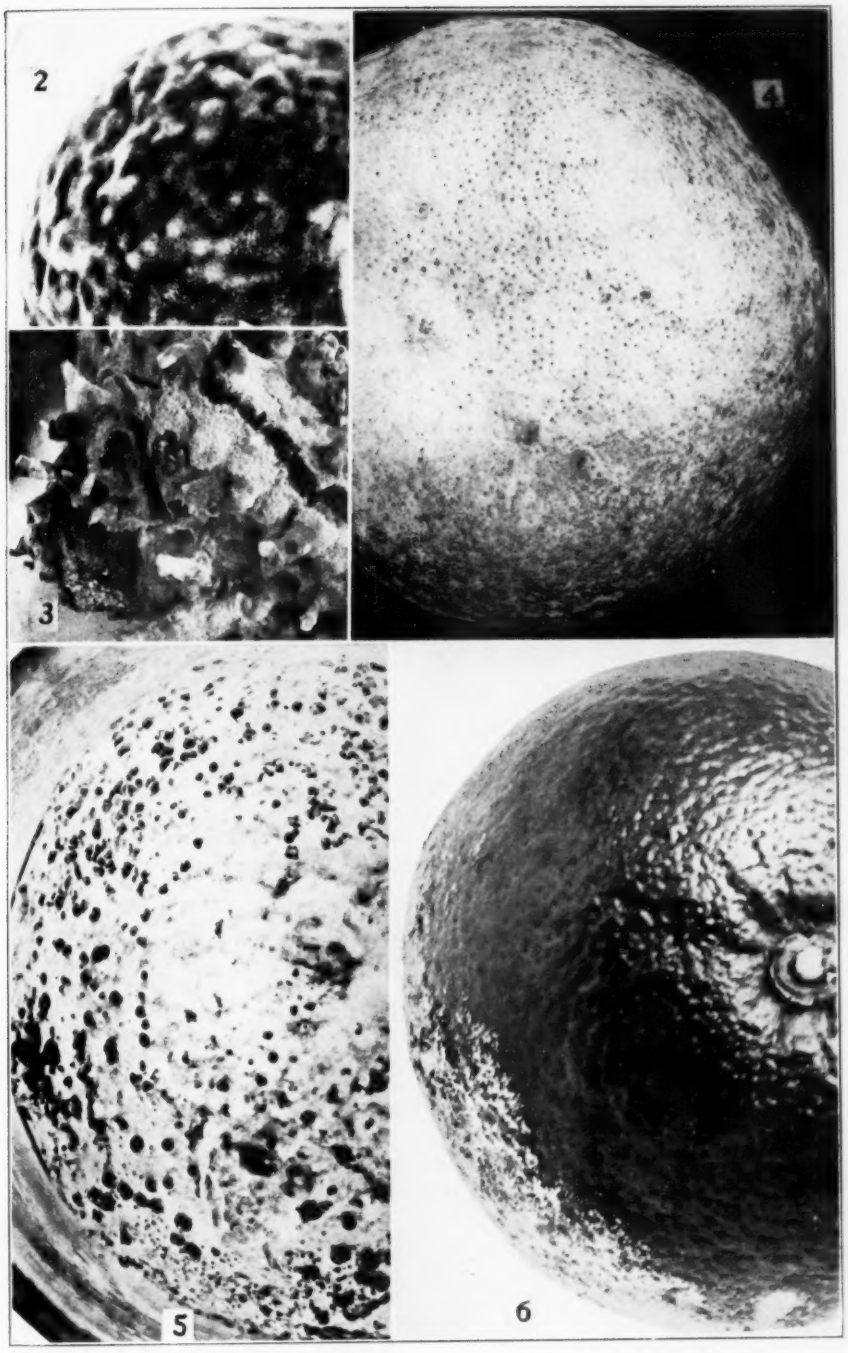
## DESCRIPTION OF PLATES XXVI AND XXVII

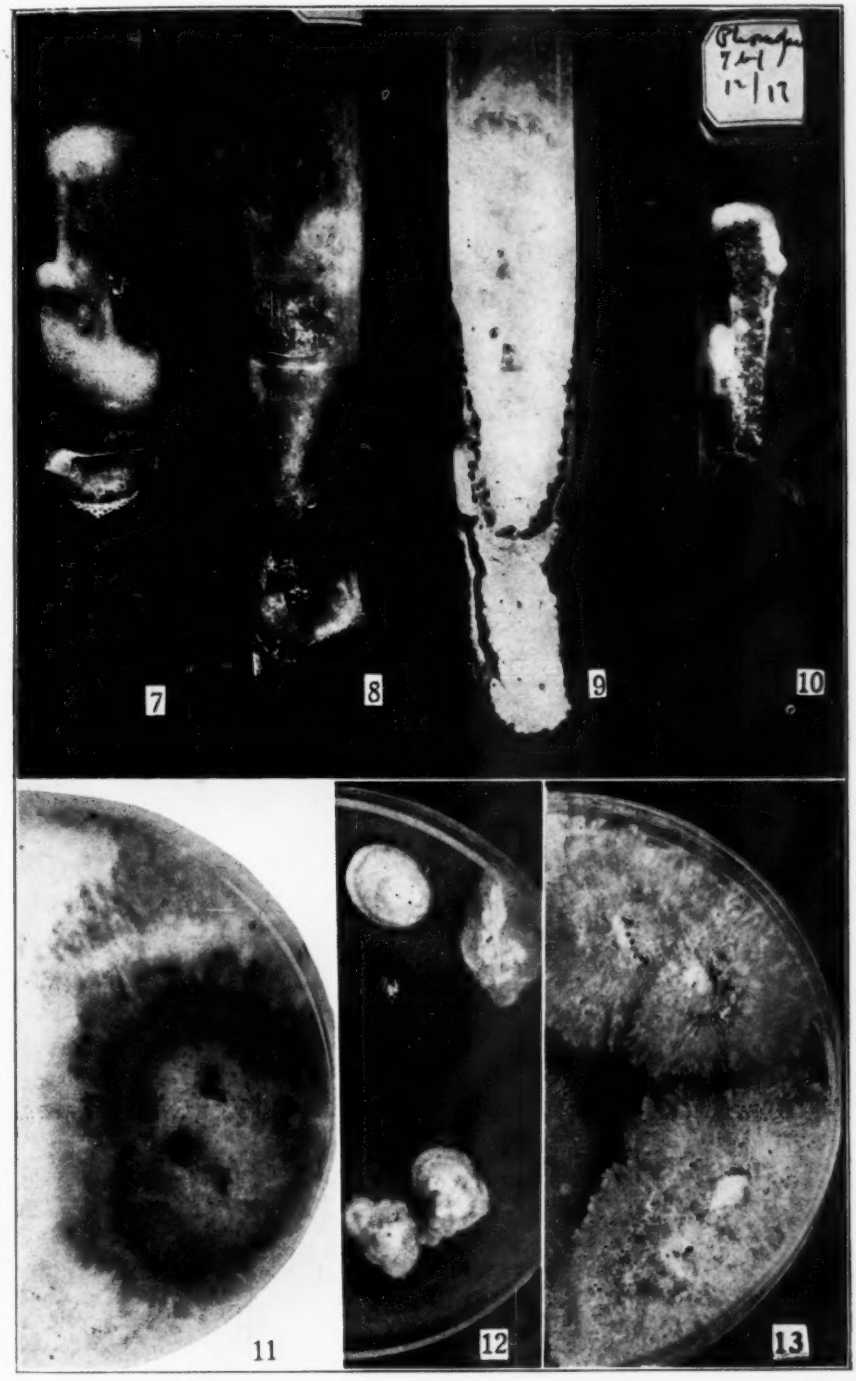
Pl. XXVI (2) Part of orange with Diplodia rot in moist chamber after dark mycelium has emerged and covered the fruit. Artificial inoculation with fungus from the Isle of Pines, W. I. (3) Very old phase of Diplodia rot in moist chamber showing sclerotial bodies, artificial culture as figure 2 on grape fruit. (4) Grape fruit from the Isle of Pines, W. I., with small spots suspected of being melanose. (5) Phomopsis from Isle of Pines inoculated on grape fruit, very old phase of culture. (6) Grape fruit with typical stem-end rot in moist chamber from inoculation with Phomopsis from the Isle of Pines fungus tufts are beginning to emerge at the left below, after the fruit is completely invaded and turned a light tan color.

Pl. XXVII (7) Culture of Diplodia from Isle of Pines on green orange twig. (8) As 7 on nutrient agar slant. (9) Phomopsis from Isle of Pines culture on nutrient agar slant. (10) As figure 9, culture on green orange twig. (11) Reverse of a petri dish culture of Diplodia from Isle of Pines in nutrient agar. Photograph pale, the black color is very pronounced. (12) As figure 9, culture in nutrient agar made somewhat alkaline in petri dish. (13) As figure 12, but medium somewhat acid.




PHOMOPSIS OF GRAPEFRUIT.

UNIV. OF MICH.




PHOMOPSIS OF GRAPEFRUIT.

UNIV. OF MICH.

# A NEW PHOMOPSIS OF CITRUS IN CALIFORNIA[1]

HOWARD S. FAWCETT

WITH TWO FIGURES IN THE TEXT

The first record of the infection of California citrus fruits by Phomopsis was made by Burger (1) in July 1919 on a shipment of lemons grown in Santa Barbara County. The seriousness of the citrus disease, known in Florida as stem-end rot, which, as shown previously by the writer (3), (2), is caused by the fungus *Phomopsis citri* Fawcett suggested further investigation.

In September 1919 the writer found a small number of fruits in packing houses in Santa Barbara County with a stem-end rot similar to that formerly described in Florida. From these fruits, a Phomopsis was isolated in culture. This decay, however, appeared to be of minor importance affecting very few fruits and only fruits that were quite mature when picked or that had been in the packing house for a long time. Since in the early stages this decay somewhat resembles other decays in lemon fruit due to *Botrytis cinerea*, *Sclerotinia libertiana* and *Diplodia* sp., it has probably been previously overlooked. The decay is usually lighter in color than the others mentioned, but can be distinguished only after some experience with it.

No fruit with stem-end rot was found in the orchards but after considerable search Phomopsis pycnidia were found on a few dead twigs, and suggestions of melanose (an important manifestation of Phomopsis attack in Florida) (5) were found on leaves. Some mature fruits picked from branches containing dead twigs and kept in the laboratory developed stem-end rot in about five weeks.

From a preliminary examination of cultures, the writer was inclined at first to think the fungus was *Phomopsis citri*. In April 1920, another search in Santa Barbara County revealed only a trace of the decay in one packing house. In the orchards after much search only a few pomelo fruits in one locality could be found with what appeared to be inconspicuous melanose markings. Later a Phomopsis similar to the Santa Barbara one was found by Dr. Bartholomew in some cultures taken from lemon fruits grown in Whittier, Los Angeles County. More recently the fungus has been found commonly by the writer in the bark

[1] Paper No. 99 University of California Graduate School of Tropical Agriculture and Citrus Experiment Station, Riverside, California.

of lemon trees affected with "shell bark," (4) and inoculation experiments have indicated that it may be a factor in the development of the shell bark disease.

## INOCULATION EXPERIMENTS

Laboratory inoculation tests to compare the California Phomopsis with *Phomopsis citri* from Florida were made in January, 1920, at a temperature between about 15° and 20° C. On lemon fruits it took 32 days to produce the same effect with the California Phomopsis as was produced by the *P. citri* from Florida in 19 days. On immature pomelo fruits the Californi Phomopsis failed to produce rotting while the Florida fungus produced the decay in 19 days.

In another test started in May, 1921, the Florida Phomopsis initiated decay at 24° C. in mature lemon, orange, and pomelo fruits in 8 days and rotted entire fruits in 14 days, while under the same conditions the California Phomopsis failed to produce decay until after the lapse of two months.

In a later test (September 29, 1921) the California fungus failed in 18 days to produce decay in very mature Valencia oranges or on nearly mature lemons (two fruits each), at temperatures of 17, 20.5, 24.5, and 27° C. Inoculations were made by placing spores and mycelium on the stem end without puncturing. After the 18-day period all the fruits were left at about 20° C. It was not until 23 days later that Phomopsis rot was noted in one lemon each of the lots started at 17 and 27° C. Only after a third period of 45 days or 81 days from the beginning was Phomopsis decay noted in about half the oranges irrespective of the original temperatures at which they had started.

## CULTURAL CHARACTERISTICS

The two fungi could readily be distinguished when grown on certain culture media. On glucose potato agar at about 20° C., in 20 days the California fungus showed a coarser growth with a tendency to form strands and fans with spaces between. The mycelium as a whole also tended to become light tawny with age and to turn to a cinnamon brown where it came in contact with this medium. The pycnidia were more abundant and tended to form only paraphyses and very few true spores. The mycelium of the Florida fungus, *Phomopsis citri*, on the other hand tended to remain pure white with a finer growth. The pycnidia was filled with true spores and contained few or no paraphyses.

On cornmeal agar the mycelium of the California fungus tended to turn to a warm buff while that from the Florida fungus remained pure white.

On sterilized orange twigs the California fungus produces numerous pycnidia with both true spores and paraphyses while *Phomopsis citri* tended to produce true spores only and no paraphyses under the same conditions.

On sterilized bean pods the pycnidia were more abundant than with *P. citri*. The true spores were abundant with both fungi, but the California fungus produced numerous paraphyses while *P. citri* showed none.

The growth of the two fungi was also compared at different temperatures, and some of the results are shown in table 1.

The growth of the California Phomopsis was found to have somewhat different temperature responses from that of the Florida fungus. (Tab. 1, and Figs. 1 and 2.)

TABLE 1

*Growth of Phomopsis californica and Phomopsi citri compared in terms of diameter of mycelium disks grown on soluble starch-glucose agar.*

| MAINTAINED TEMPERATURE DEGREES C. | ORGANISM | NO. OF CULTURES | DAYS 2 | 5 | 8 | 11 |
|---|---|---|---|---|---|---|
| | | | mm | mm | mm | mm |
| 9 | P. californica | 2 | 0 | 2.0 | 2.9 | 6.6 |
| | P. citri | 2 | 0 | 2.5 | 3.7 | 8.6 |
| 14 | P. californica | 5 | 1.3 | 7.7 | 14.7 | 27.0 |
| | P. citri | 3 | 0 | 9.0 | 13.5 | 18.5 |
| 17 | P. californica | 5 | 4.1 | 16.2 | 30.3 | 39.9 |
| | P. citri | 3 | 4.5 | 13.3 | 22.1 | 31.1 |
| 20.5 | P. californica | 5 | 6.3 | 25.8 | 37.5 | 50.7 |
| | P. citri | 3 | 7.3 | 17.3 | 29.0 | 36.8 |
| 24 | P. californica | 5 | 5.1 | 18.2 | 30.2 | 41.2 |
| | P. citri | 3 | 8.2 | 22.0 | 31.6 | 42.7 |
| 27 | P. californica | 5 | 3.5 | 7.2 | 11.0 | 14.7 |
| | P. citri | 3 | 6.5 | 18.5 | 27.5 | 38.0 |
| 30.5 | P. californica | 5 | 2.1 | 4.7 | 7.6 | 10.0 |
| | P. citri | 3 | 7.0 | 13.5 | 20.5 | 26.7 |
| 34 | P. californica | 5 | 1.4 | 1.5 | 1.5 | 1.5 |
| | P. citri | 3 | 2.5 | 3.2 | 3.5 | 4.0 |
| 37 | P. californica | 1 | 0 | 0 | 0 | 0 |
| | P. citri | 1 | 0 | 0 | 0 | 0 |

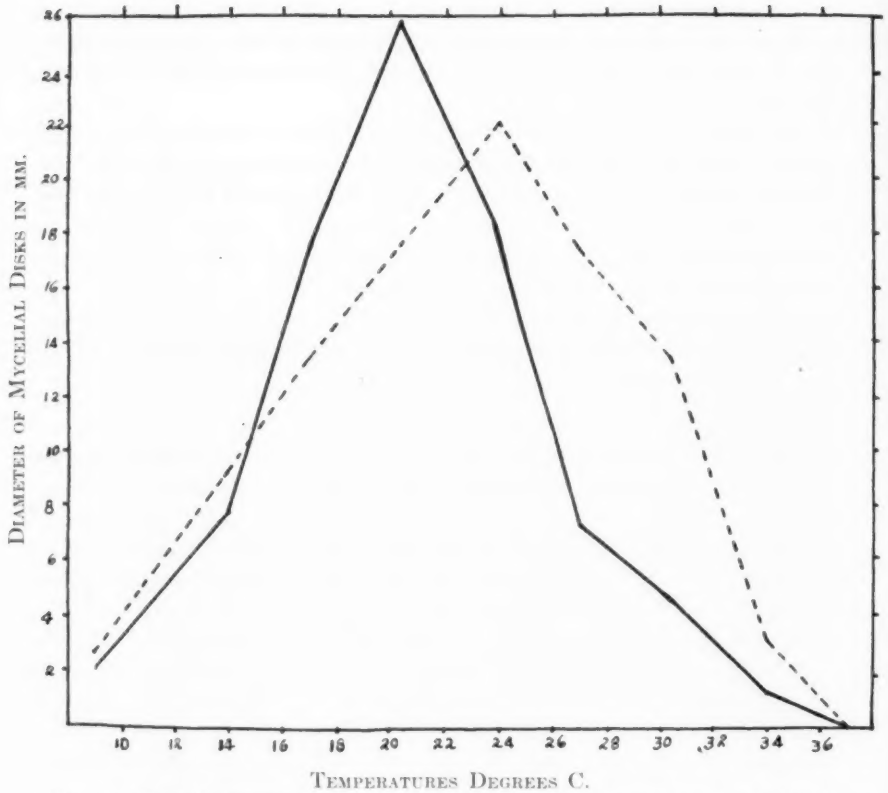



FIG. 1. Growth-temperature graphs for the first five day period on soluble starch medium (see table 1)

*Phomopsis californica* n. sp.——

*Phomopsis citri* Fawcett.- - - - -

On soluble-starch medium with one-half per cent sugar added, the California Phomopsis showed at the end of 4 days the greatest total growth at 20.5° C. while *Phomopsis citri* from Florida showed greatest growth at 24° C. The growth of the Florida fungus was also greater than the California Phomopsis, at all the temperatures tested above 24° C. The mycelial growth of the two fungi differed considerably, especially at higher temperatures. Both showed almost pure white growth initially at nearly all temperatures. The mycelium of the California fungus however, tended to become darker with age, especially at high temperature, and as a rule forms a more irregularly lobed margin. The California Phomopsis formed a thinner growth, less compact and less matted than that from Florida, at most of the lower temperatures.

The California fungus grown at 24.5° C. on soluble-starch agar for 10 days (Fig. 2, c) showed an irregular fern-like growth extending outward on the margins in the form of small separated fans with spaces between which tended to fill it later. *Phomopsis citri* (Fig. 2, e) with this same temperature and on the same medium showed a finer, more compact growth with more uniform distribution and more regular margins. At 27° C on this medium the California fungus (Fig. 2, d) grew very slowly as compared with *Phomopsis citri;* the latter grew quite well, forming a still finer more evenly distributed growth than at 24.5° C. (Fig. 2, f).

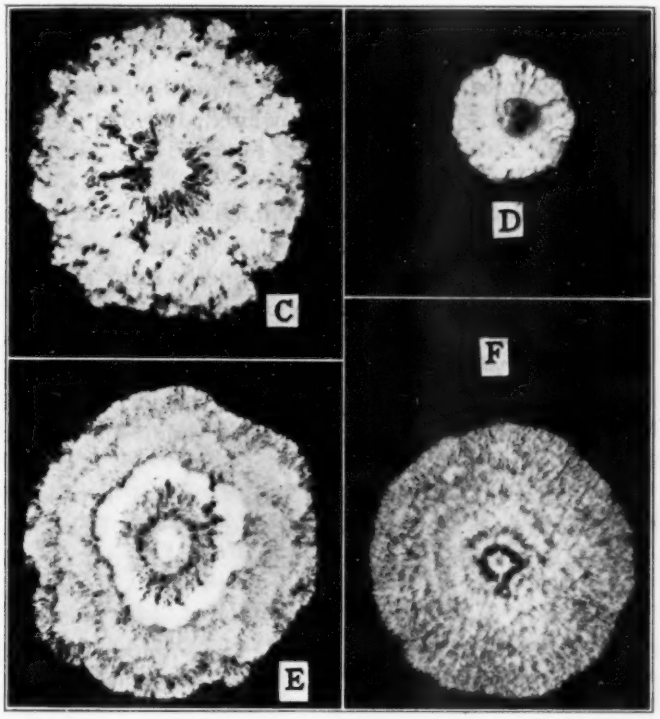



FIG. 2. (c) *Phomopsis californica* grown at 24.5° C. (d) Same fungus at 27° C. (e) *Phomopsis citri* grown at 24.5° C. (f) *P. citri* at 27° C. Natural size on soluble starch nutrient agar in 10 days.

The measurement of 100 spores of *P. californica* gave a mean length of 8.4±0.7 μ (range 7.7 to 9.1) and a mean breadth of 3.9±0.5 μ (range 3.4 to 4.3). A hundred paraphyses (sclerospores) gave a length of 22.0± 0.3 μ, the range being 21.7 to 22.5 μ. The width was about 1.6 μ. These spore measurements are nearly like those of *Phomopsis citri* grown

under the same conditions, the measurements of 100 spores for the latter species giving 8.4±0.6 μ for the length (range 7.8 to 9.0) and 3.8±0.5 μ for the breadth (range 3.3 to 4.2). The two cannot therefore be separated on the measurements of the spores alone.

The weaker virulence of the California Phomopsis together with its lower optimum temperature for growth, difference in mycelial growth and difference in spore production (paraphyses rather than spores being the predominating characteristic on a variety of culture media), indicates that the fungus should probably be considered a different species from that commonly found in Florida. It is highly probable that the fungus occurred in California for a long time and has been previously overlooked because of its slight importance in producing decay. The following description may be added:

**Phomopsis californica** n. sp. Pycnidia mostly clustered, sometimes rarely scattered, ovoid to conical, dark colored, 160–300 μ in diameter, erumpent. Ostiole 25–35 μ in diameter. Spores ovate, mostly rounded at both ends, sometimes nearly acute at one end, hyaline. Mean size of 100 spores measured, 8.4±0.7 μ long and 3.9±0.5 μ wide. Range 7.7–9.1 x 3.4–4.3 μ. Basidia about 15 μ long, paraphyses (sclerospores) 22–0.3 x 1.6 μ, curved at one end. Differs distinctly from *Phomopsis citri* Fawcett in coarser hyphae with tendency to form strands or fans of growth, the hyphae becoming cinnamon brown instead of pure white when in contact with glucose potato agar; also differs by forming principally paraphyses instead of spores on above media. On dead outer bark and in decaying fruits of *Citrus limonia* in California, United States of America.

Specimens of the fungus on lemon bark and on several different culture media are being sent to the herbarium of the Bureau of Plant Industry, Office of Pathological Collections, Washington, D. C.

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# OBSERVATIONS ON TWO POPLAR CANKERS IN ONTARIO

E. H. Moss

## I. *Dothichiza populea* Sacc. and Briard.

Poplar canker caused by *Dothichiza populea* Sacc. and Briard is a well-established and destructive disease in Europe. In France, according to Delacroix (1), it is very troublesome to newly planted trees and in nurseries, attacking varieties of *Populus nigra*, but being particularly destructive to the Carolina poplar (*P. deltoides*). In America, the canker was reported for the first time in 1916 by Hedgecock and Hunt (2). These investigators have observed the disease in several of the New England and Northeastern States, where it is said to occur chiefly on *P. nigra* but also on *P. deltoides*. As far as the writer is aware no other paper on the subject has been published in America.

The writer's attention was first drawn to this disease in the spring of 1921 when he was asked by Dr. J. H. Faull to examine some young Lombardy poplars which had been planted in 1920 by the Harbor Commission of the City of Toronto, Canada. It was found that over 90 per cent of the trees—500 in number—were girdled by a fungus, which was identified as *Dothichiza populea*. In most cases the girdled area extended over a considerable length of stem and was located about two feet from the tip. From numerous pycnidia on the diseased parts spore horns appeared, the latter being in evidence as early as May 4.

Later in May, the disease was observed at widely separated points (in Middlesex and Oxford Counties) in southern Ontario, on old Lombardy poplars. In fact, almost every clump of these poplars examined showed evidence of the disease. In most cases, young shoots from the roots of the old trees were very markedly affected. These were being killed back, the young and immature leaves being blackened, and pustules appeared in large numbers on the stems. In most of the trees several branches situated at varying heights displayed evidences of the disease. Many of the older branches were disfigured by elongated open wounds, while younger shoots were being killed back.

Hedgecock and Hunt (2) observed only a few cases of infection of older trees by this pathogen. They suggest that the fungus has been

brought to America from Europe, but that it may have been brought in "previous to the enforcement of the present inspection laws." However, owing to the fact that *D. populea* has apparently not been collected or reported by American investigators, Hedgecock and Hunt come to the general conclusion that "it is a somewhat recent disease in the United States." Of course, the fungus may have been introduced on nursery stock on more than one occasion, but the writer's observations indicate that Hedgecock and Hunt are correct in their first suggestion. In Ontario the disease has undoubtedly been prevalent on poplars for many years, and consequently it is evident that *D. populea* is not recently new to America.

### II. *Cytospora chrysosperma* (Pers.) Fr.

The canker disease of poplar and other trees which is caused by *Cytospora chrysosperma* (Pers.) Fr. is apparently widely distributed in America. It has been reported as occurring in nine southwestern and central States by Long (4), in Idaho, Washington and Wyoming by Hubert (3), and in New York State by Povah (5). The present writer has observed the disease at various places in the neighborhood of Toronto and at Guelph, Ontario. The host most commonly attacked and most susceptible to the fungus in Ontario seems to be *Populus deltoides*. In addition, *Populus italica*, *P. balsamifera*, *P. alba* and *Acer saccharinum* have been observed as hosts. The latter is here reported as a new host for this pathogen.

According to Long, when *P. alba* is attacked by this fungus, the affected trees die branch by branch from the top downward. The writer has seen trees of *P. alba* and also Lombardy poplars (*P. italica*) which were being parasitized and slowly killed in this manner.

In a certain district in the vicinity of Toronto, it was observed that numerous young trees of *P. deltoides* were infected. Pycnidia of the fungus occurred near wounds in the younger branches and also on the lower parts of the trunks. As a result of these trunk infections some of the trees were being rapidly killed. It was apparent that the ground had been burned over and that the pathogen had attacked the trunks following fire injury. This observation is in accord with earlier accounts (3, 5) of this disease on poplars injured by fire.

### SUMMARY

Observations made during 1921 and 1922 make it clear that *Cytospora chrysosperma* and *Dothichiza populea*—two canker-producing wound parasites of the poplar—are not of uncommon occurrence in southern

Ontario. Furthermore, it would seem that *D. populea* has existed on this continent for a considerable length of time and that it has not been introduced from Europe as recently as has been suggested elsewhere.

Older trees of certain species of Populus commonly planted are rendered unsightly and are gradually killed by these fungi. Younger trees of *P. deltoides*, especially if seriously injured or weakened, are likely to succumb to the attacks of *C. chrysosperma*, and infected nursery stock of *P. italica* is rapidly killed by *D. populea*.

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UNIVERSITY OF ALBERTA.

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# THE SPREAD OF TOMATO WILT BY INFECTED SEED

JOHN A. ELLIOTT AND R. F. CRAWFORD

WITH PLATE XXVIII AND TWO FIGURES IN THE TEXT

The first suggestion that tomato wilt is a seed borne disease was made by Clinton (1, 2) from the circumstantial evidence that the amount of wilt increased from year to year when seed from infected plots was saved for planting. No definite proof that seed carried the fungus was given by Clinton, but his suggestion has apparently been cited as established proof by Norton and Leathers (5). In his bulletin on the development of wilt resistant tomatoes, Pritchard (6) states that the disease is seed borne, but records no experiments establishing the fact and makes no citation of other work. His statement is as follows:

"Wilt is carried to some extent by the seed, but not so commonly as the high percentage of fruit infections in wilt-infested fields would seem to indicate. The fungus passes through the fibro-vascular bundles of the fruit to the seed and often invades the cells surrounding the seed coat. Were it not for the removal of these cells through fermentation and washing of the seed in the seed-saving process the infection of plants through the seed would be much more common. The fungus-bearing particles separated from the seed by fermentation frequently adhere to it, however, and thus become a source of infection for the plant and a means of more widespread distribution for the fungus. Infection of plants through the seed would be more common if tomato seed was produced commercially in badly wilt infested regions."

The nearest approach to proof that tomato wilt is seed borne, appearing in literature, is Edgerton and Moreland's (3) account, which might be said to establish the possibility that the disease can be carried on the seed. Edgerton and Moreland infected tomato seed with a pure culture of the wilt organism (*Fusarium lycopersici*) and succeeded in growing wilt infected plants from the infected seed the following planting season, proving that the fungus could live on the seed from the time the seed was collected to the planting time of the next year's crop in the region where they worked, a period of over three months.

Tomato wilt is the limiting factor in the successful production of tomatoes in Arkansas. In a large part of the state the disease is so generally present that no large plantings are undertaken, and even garden production of tomatoes for home use is attended by uncertainty

in most parts of the state. While so general a distribution of the disease might be expected in time from the introduction of the fungus on diseased plants, this explanation of its introduction into many fields, as has been pointed out by Edgerton and Moreland (3), is unsatisfactory. In Northwest Arkansas in particular, where the tomato canning industry has made a rapid growth and tomatoes are often grown from seed planted directly in the field, the general distribution of the disease in many such fields the first year they are used for tomato growing seems inexplicable on any other grounds than the introduction of the disease with the seed. This was so apparent in some virgin fields in 1920 and 1921 that very careful selection of seed from badly wilted plants in which the fungus had thoroughly invaded the fruit, was made in September and October, 1921, with the object of ascertaining whether such seed carried the wilt fungus.

The seed was cleaned by fermentation and washing, then dried and put away in an Erhlenmeyer flask plugged with cotton. The seed had a very clean bright appearance, but contained a considerably greater amount of small seeds than is usually found in commercial stock. The seed received no further attention until January 20, 1922, when planting was begun.

Since it was desired to isolate the wilt fungus only, the methods of isolation were designed to accomplish that end, and several modifications of the usual technique were tried. Very little plating was done by the poured plate method, but sterilized blotting paper was used instead. At first the seeds were placed, after various treatments, directly on the blotting paper; later, black paper was placed over the blotting paper and the seeds distributed over the surface of the black paper. Any fungous growth was very readily seen against the background of this black paper (Fig. 1). Fungi appearing on the seeds were examined microscopically and any which appeared to be the wilt fungus were saved for identification. The final test of identity as *F. lycopersici* was in all cases the production of typical wilt in tomato plants. However, the identity was established to a practical certainty by preliminary morphological and cultural tests. Any fungus which had the appearance of the wilt fungus when examined under the microscope was transferred to tubes of boiled rice, where, if it was *F. lycopersici*, it gave a very characteristic color reaction, which in our experience has always proved to be dependable (7). As nearly as it can be described from Ridgway's Color Standards and Nomenclature[1], the color produced on rice was alizarine pink in

[1] Ridgway, Robert. Color Standards and Nomenclature. Published by the author. Washington, D. C., 1912.

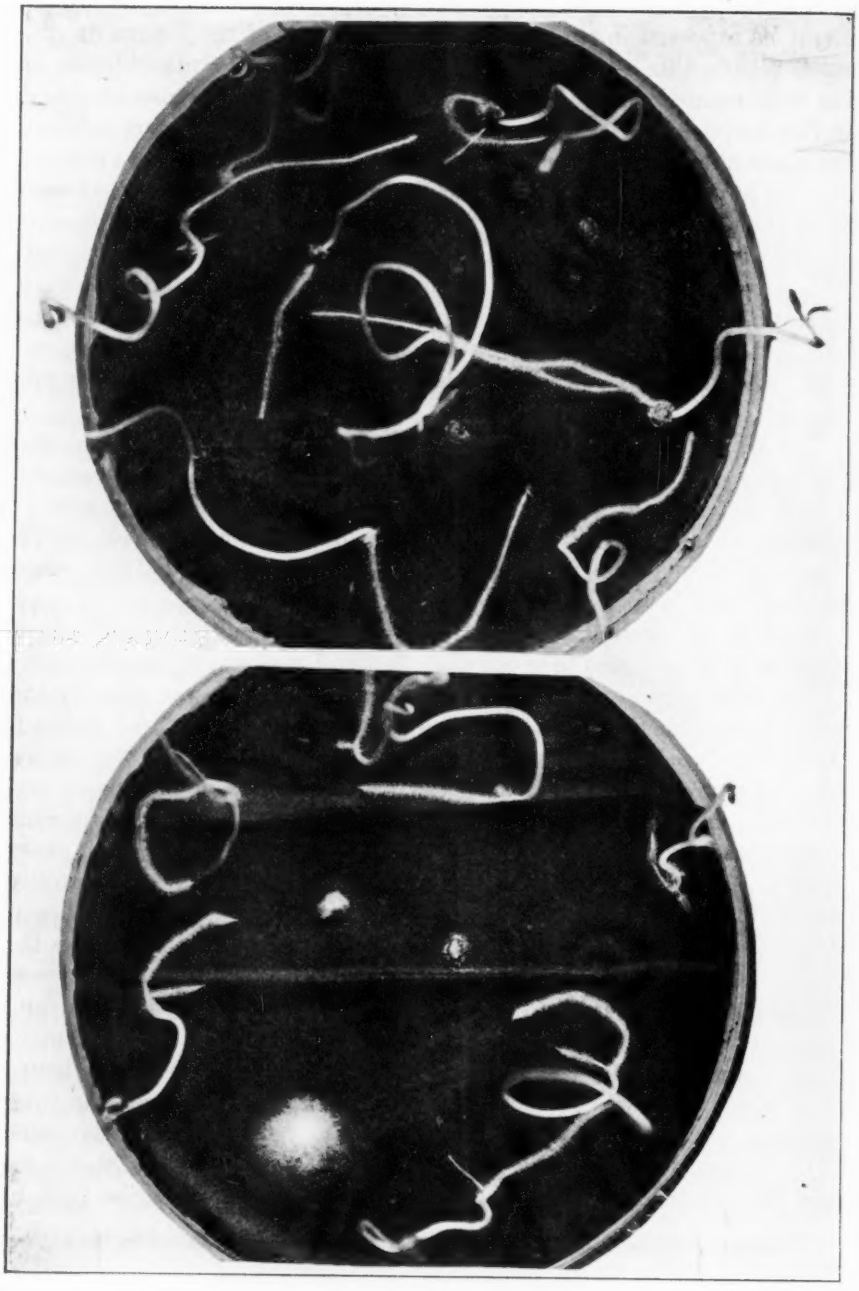

FIG. 1. Tomato seed from wilt infected tomato plants plated on sterilized black paper. Above, all seeds healthy. Below, 1 seed showing growth of *F. lycopersici*.

the lighter portions, and old rose, or Begonia rose, in the darker portions of the colony. The color perhaps approached a little nearer pure red than these, but no other colors given are so near it. All cultures giving this characteristic color reaction on rice produced wilt in tomato plants in subsequent tests.[1] Another modification in technique which was found useful in indicating the wilt fungus while growing on the blotting paper, was suggested by the color reaction given on rice. By dipping the white blotting paper in rice water before auto-claving the culture plates, the wilt fungus was made to produce a color reaction on the original plates, which aided in picking it out even when the growth of the wilt fungus was masked by some other fungus growing from the same seed (Pl. XXVIII). On the rice-starch-blotting-paper the fungus produced a deep red-violet color somewhat suggestive of the iodine starch reaction.

Three methods of treating the seed, previous to plating, were employed, as follows: 1. Seed was placed in sterile water in order to be easily separated, then transferred to the sterilized paper without any other treatment. 2. Seed was soaked for 2 minutes in 1–1000 mercuric chlorid solution, washed in sterile water and transferred to plates. 3. Seed was surface charred in concentrated sulphuric acid, washed in water, soaked for 2 minutes in 1–1000 mercuric chlorid solution and plated. Aside from Penicillium, which was evidently accidental contamination, and an occasional Aspergillus, the only fungus appearing on the plates,

TABLE 1

*Fusarium lycopersici isolated from tomato seed.*

| SERIES | DATE | TREATMENT | NO. SEED | ISOLATIONS OF F. LYCOPERSICI | ISOLATIONS LIVING SEED | FROM DEAD SEED |
|---|---|---|---|---|---|---|
| A | 1/20/22 | Soak in $HgCl_2$. 2 min. | 100 | 1 | — | — |
| B | 1/20/22 | None | 100 | 2 | — | — |
| C | 2/ 4/22 | Soak in $HgCl_2$. 2 min. | 100 | 1 | 1 | 0 |
| D | 2/ 4/22 | None | 100 | 3 | 0 | 3 |
| E | 3/14/22 | $HgCl_2$ 2 min. | 100 | 0 | — | — |
| F | 4/17/22 | None | 100 | 6 | 0 | 6 |
| G | 5/ 4/22 | Sulphuric acid, and $HgCl_2$ 2 min. | 240 | 1 | 0 | 1 |
| H | 5/11/22 | None | 100 | 2 | 0 | 2 |
| I | 5/11/22 | $HgCl_2$ 2 min. | 100 | 0 | — | — |
| J | 5/11/22 | Sulphuric acid, $HgCl_2$ 2 min. | 150 | 3 | 0 | 3 |

[1] This same test was made in the case of the cotton wilt fungus, with which a practically identical color reaction was obtained.

FIG. 2. Tomato plants about three months old dying of wilt in pots inoculated with *F. lycopersici*. Right, pot inoculated with a known wilt culture. Pots at left inoculated with isolations A1, and B1. Other plants had died earlier.

aside from the wilt organism, was *Mucor sp.* which apparently came from the seed. This was true of all the seed plated, regardless of previous treatment. The most striking thing was that even the untreated seeds were practically sterile as far as fungus growth was concerned. The method of handling the seed gave little opportunity for the development of bacteria even if they were present. The results of the plantings are given in table 1.

The difference in the number of isolations of the wilt fungus from the surface sterilized and the untreated seed would indicate that the organism is carried on the outside of the seed coat, as a rule. More than 80 per cent of the seed germinated except when treated with sulphuric acid, when practically none germinated. At first no record was kept of the number of isolations made from dead or from viable seeds, but most of them came from dead seeds. The isolation of the fungus from the seed treated with sulphuric acid would indicate an internal infection, as it would seem impossible for the fungus to survive this treatment if it was not beneath the seed coat.

## INFECTION EXPERIMENTS

The pathogenicity of the suspected fungi was tested by the method devised by Edgerton (4). Autoclaved pots of soil were inoculated with pure cultures of the fungi to be tested, by means of infected fragments of cotton stems, and the tomato seed planted at the same time. Check pots of known wilt fungus, and also uninoculated pots were run in the same series. Plants in the inoculated pots began to show infection in about a week and died of wilt from then on. Some of the plants lived for three months or more (Fig. 2). In the smaller plants the dark streaks due to infected vascular bundles could be seen without cutting the stems. No infections occurred in the uninoculated pots. Considerable

TABLE 2

*Inoculations with F. lycopersici isolated from tomato seed.*

| ISOLATION SERIES | INOCULATED | 1ST PLANT SHOWING WILT | | |
|---|---|---|---|---|
| A 1 | 2/20/22 | A 1 | 3/24/22 | |
| B 1, 2 | 2/20/22 | B 1, 2 | 3/24/22 | |
| C 1 | 3/27/22 | C 1 | 5/ 5/22 | |
| D 1, 2, 3 | 3/27/22 | D 2, 3 | 6/ 1/22 | D 1 6/5/22 |
| F 1, 2, 3, 4, 5, 6 | 6/ 1/22 | F 5 | 6/ 5/22 | F 1, 2, 3, 4, 6 6/16/22 |
| G 1 | 6/ 1/22 | G 1 | 6/12/22 | |
| H 1, 2 | 6/ 1/22 | H 1, 2 | 6/16/22 | |
| J 1, 3, 4 | 6/ 2/22 | J 4 | 6/16/22 | J 1, 3 7/1/22 |

difference in the virulence of different isolations of the fungus was evident. In some pots the plants died very rapidly while in others they survived much longer. This difference in virulence of different cultures of *F. lycopersici* has been previously reported by Edgerton and Moreland (3). The results of the inoculation experiments are shown in table 2.

How long the fungus will remain viable on the tomato seed remains to be determined. These experiments show that naturally infected seed may carry the viable organism for at least seven months, a period long enough to carry the disease over from one season to the next in any tomato growing section of the United States. The seed was very carefully selected from fruits most heavily invaded by the wilt fungus but still the percentage of seeds from which the fungus was isolated was surprisingly high, being 3½ per cent in the case of the untreated seed, which should give the best index to the amount of wilt that seed from badly infected plants may be expected to carry.

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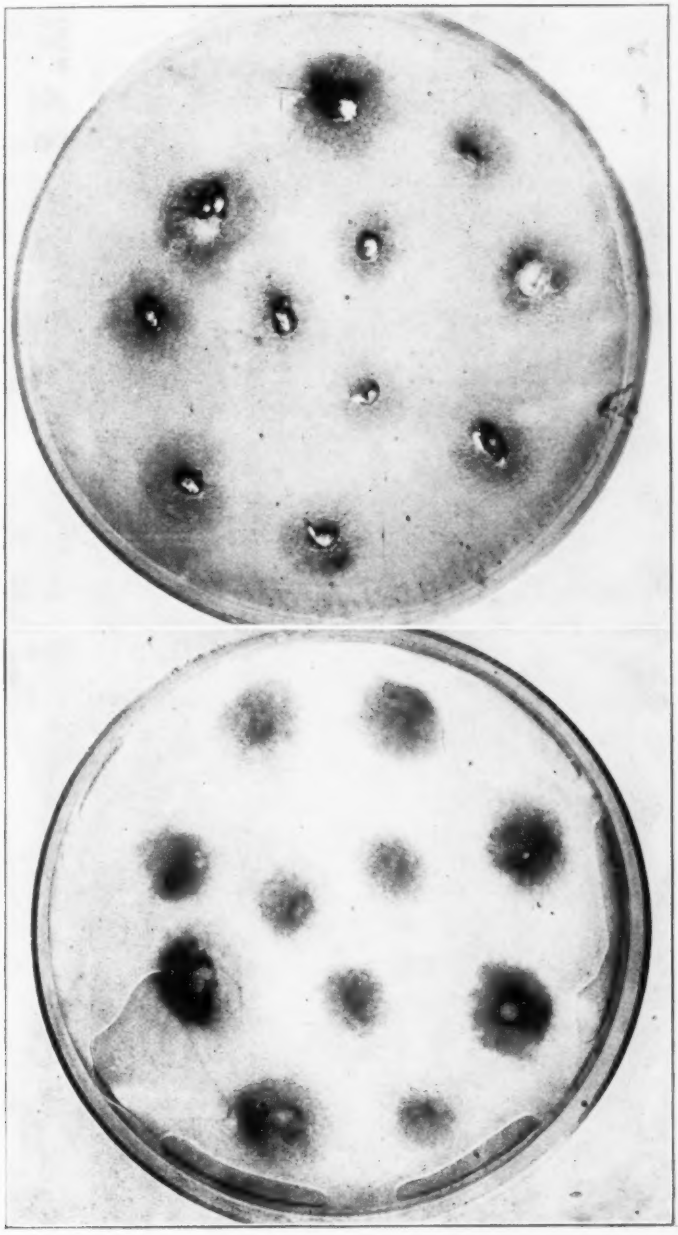

TOMATO WILT

Tomato seeds artificially inoculated with spores of *F. lycopersici* plated on autoclaved rice-starch-paper, showing color reaction after 48 hours. Above, upper side of plate; below, lower side of same plate.

UNIV. OF MICH.

# A NEW SPECIES OF SCHIZONELLA

M. O. Parthasarathy Iyengar and M. J. Narasimhan

With Four Figures in the Text

This fungus was found growing on *Vitis quadrangularis* Wall. It causes a sort of witches' broom like growth at the nodes which at first sight might easily be mistaken for a kind of Viscum growing on the plant (Fig. 1). The diseased growth is very much unlike any part of the normal plant. These diseased parts start out at the nodes and branch repeatedly in all directions in a very irregular fashion so that the

Fig. 1. *Vitis quadrangularis* Wall. with the diseased growth on it caused by *Schizonella colemani*.

total growth at the node has more or less a spherical shape. These branches are pale green in colour with a reddish tinge towards the ends of the branches. A good number of dark green longitudinal swellings is found on these branches. Each of these green swellings when cut open shows a large quantity of dry, black, powdery spores lying loose inside. These green swellings are quite small near the ends of the

branches but become larger in size as the branches grow older and longer. The length of the swellings in the older branches is very nearly half an inch (Fig. 2).

SPECIES HITHERTO RECORDED (3)

1. *Schizonella subtrifida* on *Cirsium ochrocentrum:* spore 12–20 μ by 12–16 μ. American species.

2. *Schizonella melanogramma* on Carex spp.: spore 8–12 μ by 5–8 μ. American species.

3. *Schizonella melanogramma* var. *elynae* on *Elyna spicata:* spore 6 μ. Norwegian species.

FIG. 2. A few branches with one of the longitudinal swellings cut open showing the black spores inside.

DESCRIPTION OF THE MADRAS SPECIES

**Schizonella colemani,** n. sp.

Spores coupled in pairs through a narrow isthmus, each half being approximately semi-circular, the two together forming a sort of a dumb-bell (Figs. 3, 4). Epispore dark brown, smooth. Spore measurement 14–16 μ from end to end of the double spore by 7.9–10 μ across, being the average of ten measurements.

*Locality.* The specimen was collected once at Pallavaram and at another time at Vandalur, both stations very near Madras.

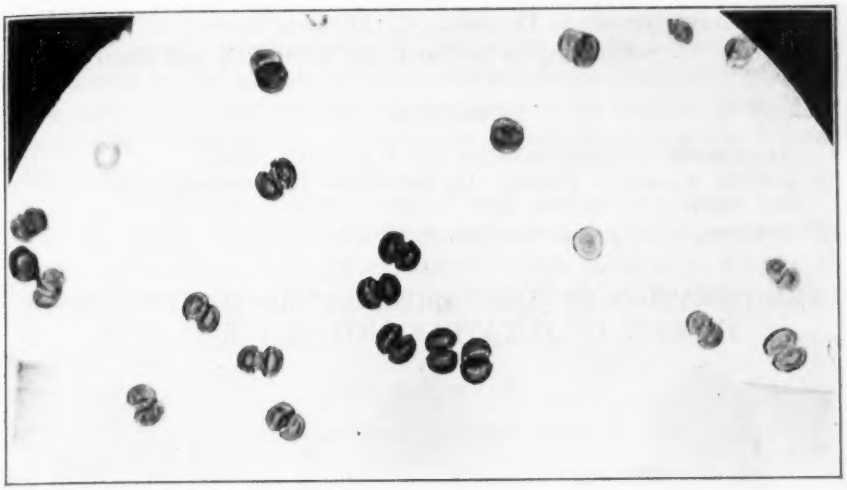

FIG. 3. Microphotograph of the double spores. C Zeiss Obj. 8 mm. OK. 12.

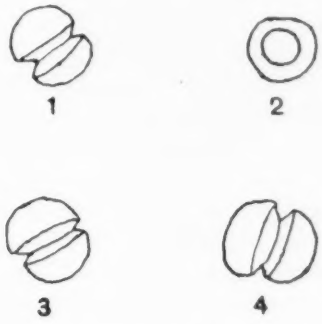


FIG. 4. Spores of Schizonella. × 660.

## GERMINATION

The spores did not germinate even though we tried hanging drop cultures in 1, sterilized water, 2, horse dung solution, and 3, malt extract. It may, however, be mentioned that the germination of the spores of the type species of the genus (*Schizonella melanogramma* D. C.) has been recorded. Rabenhorst (1) gives a figure of the germination of the spores of *S. melanogramma* and says "Das Promycel trägt die sporidien seitlich." Engler and Prantl (2 p. 12) with reference to the

spores of the same species say "Keimung wie bei Ustilago, mit hefeartiger Sprossung der Conidien." The cultural characters of the species under consideration will be studied later.

Our thanks are due to Dr. Leslie C. Coleman, Director of Agriculture in Mysore, for permitting us the use of his laboratory and library.

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# THE RELATION OF AIR TEMPERATURE TO THE MOSAIC DISEASE OF POTATOES AND OTHER PLANTS

JAMES JOHNSON

WITH ONE FIGURE IN THE TEXT

It has recently been shown by the writer that the optimum and maximum temperatures for the mosaic disease of tobacco are 28 to 30° C. and 36 to 37° C. respectively.[1] Similar experiments have since been conducted with mosaic diseases of various other plants, including potatoes, tomatoes, soy beans, pea beans, and clover, special attention being given to the potato mosaic. The method followed in potatoes has been essentially that of placing young potato plants showing symptoms of mosaic into the air control chambers, parts of the individual tubers in each series being represented in each chamber. Potato plants have been held at fairly constant temperatures for from one to three weeks at temperatures ranging between 6° and 36° C. The effect of temperature on the mosaic development was gauged by the intensity of the symptoms or the rate of "recovery" from the disease (Fig. 1).

The results have shown that the optimum and maximum temperatures for potato mosaic are considerably lower than those for tobacco mosaic. The most favorable temperature could not be determined closely since the disease is persistent at temperatures where the potato foliage makes little or no growth. Temperatures as low as 6° C. seemingly did not inhibit the disease. Taking the growth of the host into consideration, the optimum temperature lies between 14° and 18° C. Above 20° C. symptoms disappear, the rate of recovery from the dis-

[1] Johnson, James. The relation of air temperature to certain plant diseases. Phytopath. **11**: 446–458, *2 fig., pl. 21 to 23.* 1921.

ease being increased in proportion to increase of temperature within the limits of host development. To completely inhibit the disease, however, within a period of one to two weeks, a temperature of 24 to 25° C. is necessary; and this may be regarded as the maximum temperature for mosaic manifestation in the potato. New leaves, free from symptoms, appear quickly at this temperature and older leaves gradually lose their symptoms, the rate of "recovery" being roughly proportional to the age of the leaf, i. e., the older the leaf the longer the time required for recovery.

Potato plants and tubers infected with mosaic have been kept for varying lengths of time at 30° and 36° C. with the expectation that long treatment at such temperatures would eventually destroy the mosaic virus. Exposures as long as 10 days at 36° C. have failed to

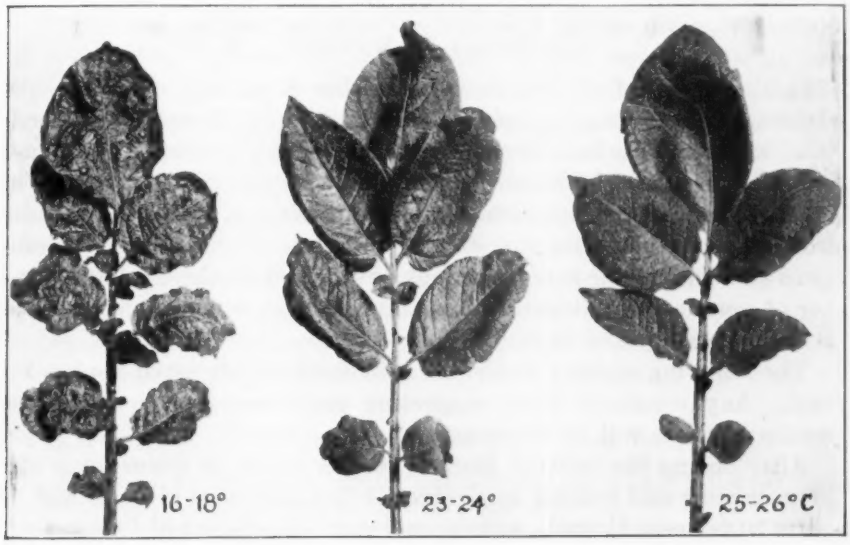


FIG. 1. *The Influence of Air Temperature On The Mosaic Disease of Potatoes.* The foliage grown at a temperature of 25–26° C. lost all symptoms of the disease in seven days. Faint symptoms were still evident at 23–24° C.

entirely destroy the virus, although some indication has been obtained that longer treatment may be effective without destroying the germination of the tuber.

The response of the potato mosaic disease to temperature is in accordance with field observations on the behavior of the disease under different environmental conditions. It is important that such temperature responses be considered in experimental work with mosaic, and in testing tubers for seed, as well as in potato inspection work, and in certification records.

Certain other mosaic diseases have been worked with in less detail. These have all responded to temperatures in a manner similar to tobacco and potato, some falling in the high temperature class with tobacco, others behaving more like the potato mosaic, and others being intermediate in this respect. Mosaic of tomato (inoculated with the tobacco mosaic virus) is for instance, most active at high temperatures, whereas clover mosaic is seemingly favored by low temperatures. Soy bean mosaic is inhibited at temperatures of from 26 to 28° C., but the pea-bean mosaic can apparently persist at a considerably higher temperature.

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## A STAINING METHOD FOR HYPHAE OF WOOD-INHABITING FUNGI

ERNEST E. HUBERT

In the course of the routine examination of various woods for the determination of decay, a rapid method for staining the hyphae of wood-inhabiting fungi has been developed. The methods developed by Diemer and Gerry (1) and by Sinnott and Bailey (2) give excellent results in differentiating the wood-rotting fungi from the host tissue but require from 12 to 24 hours for the staining process. Under ordinary conditions this time factor is not a serious drawback but where a large number of routine decay determinations are involved, a more rapid method is both necessary and desirable.

The following staining method is submitted to interested readers for trial. Any communications suggesting improvements or giving the results of trials will be appreciated.

After boiling the infected blocks (3/8 inch cubes) in water for a half hour or more and soaking in glycerin-alcohol (50 parts glycerin and 50 parts 70 per cent alcohol), sections were cut and stained as follows:

1. Drain excess water from sections. Flood with bismark brown (2 per cent solution in 70 per cent alcohol) 1 to 2 minutes according to species of wood, density, thickness of section, stage of decay, etc.
2. Drain excess stain and wash with distilled water.
3. Flood sections for from 2 to 5 minutes with a solution of methyl violet made by mixing 4 parts of a saturated aqueous solution of methyl violet with 12 parts of distilled water. In some cases use full strength for from 1 to 2 minutes.

[1] Published by permission of the Director.

4. Drain excess stain and wash with distilled water.

5. Mount in water and examine for depth of staining. If violet color is faint repeat 3 and 4 using full strength stain for from ½ to 1 minute. If counterstain is faint, repeat, beginning at 1.

6. Dry slowly on warming plate using a cover glass to keep the sections flat. If sections curl use egg albumen or gum arabic fixative.

7. Add xylol and mount in balsam.

Hyphae of wood-destroying fungi, ascomycetes, molds, sap-stain fungi, etc., stain a deep violet color. The cell walls of wood stain yellow to brown. Wood tissues containing the cellulose-complex stain slightly with methyl violet giving a mixed brown and violet color. The wood tissues having the cellulose-complex mostly removed stain yellow to brown. The contents of the medullary rays and the bordered pits in conifers usually stain a violet color.

The drying method has been used in place of the ordinary method of dehydration by alcohol. The results secured seem to justify the means. The writer realizes that the drying method is contrary to the general practice but dehydrating with alcohol apparently removes the violet stain. Other methods of dehydration may be found practicable.

This method has been successfully used in staining and bringing into vision the minute hyaline hyphae of wood-rotting fungi which in the unstained sections are ordinarily invisible under the microscope. It has been tried out on a representative number of wood-inhabiting fungi and so far has given satisfactory results.

INVESTIGATIONS IN FOREST PATHOLOGY,
BUREAU OF PLANT INDUSTRY,
IN COOPERATION WITH THE
FOREST PRODUCTS LABORATORY, MADISON, WISCONSIN.

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## *ABSTRACTS OF PAPERS PRESENTED AT THE SIXTH ANNUAL MEETING OF THE PACIFIC DIVISION, AMERICAN PHYTOPATHOLOGICAL SOCIETY, SALT LAKE CITY, UTAH, JUNE 22 TO 24, 1922.*

*Fig smut studies.* ELIZABETH H. SMITH AND EDITH H. PHILLIPS.

In an investigation of the fig "smut" problem in California the following points were established by the writers during the fall of 1921. This refers to the infection of white varieties of fig fruit by *Aspergillus niger* v. Tiegh.

Time of infection. Infection was found to occur chiefly on the tree after the fruit is mature enough to be succulent but before much shriveling takes place. Infection may occur in firm, green figs. Development is most rapid just before the stage when picked for marketing fresh, and is entirely checked as soon as the pulp becomes brown and adhesive. This was proved by careful and extensive culture and inoculation experiments with figs of all stages on and under the tree.

Type of infection. The rot is firm, dirty white. It usually starts from the eye end and involves both skin and pulp in unshriveled figs, the surface remaining clean except in contacts. Pockets lined with the characteristic black spore masses are finally formed in the pulp.

Mode of infection. Mode of infection is almost entirely by the entrance of spores through the eye. Insects and smut spores were found in small hard figs, still sound, with closed eyes. Preliminary experiments indicate that insects, rather than wind, are the chief carriers.

*Notes on Synchytrium.* JAMES MCMURPHY.

*S. papillatum* Farlow has recently been found on *Erodium moschatum* L' Her. A *Synchytrium* with spores 200 to 240 $\mu$ long in multicellular galls has been found on three species of Lotus (Hosackia).

*Morphological differences between Nectria galligena Bres. and N. coccinea (ditissima).* S. M. ZELLER.

*Nectria galligena* which has been described by Bresadola as the organism causing the European canker of apple and pear, is distinct from *Nectria coccinea* Fries (*N. ditissima* Tul.) to which until recently the cause of the canker has been attributed. These two species differ in their morphology as well as in their parasitic virulency and physiology. The perithecia differ in structure. In perithecia of *N. coccinea* found on various native hosts in Oregon the walls are made up entirely of pseudoparenchyma up to the ostiole, while in *N. galligena* from the same districts the pseudoparenchyma in the walls of the perithecia extends up about three-fourths the distance from the base. The remainder of the perithecial wall is of long, narrow cells, which radiate from the ostiole forming a cone.. The perithecia of the two species are practically the same size and color. The ascospores of *N. galligena* are 14 to 22 $\mu$ in length, while those of *N. coccinea* are 9 to 14 $\mu$. In Oregon collections the conidia (*Fusarium willcommii*) of *N. galligena* have the average measurement of 65.9 by 4 to 5 $\mu$ and those of *N. coccinea*, which are also a *Fusarium* spore, are 54 by 6 $\mu$. The latter have more rounded ends and have a curvature of shorter radius than those of *N. galligena*. The conidial stromata of *N. coccinea* are orange colored, while those of *N. galligena* are creamy-white.

*A "plum pocket" on Prunus subcordata in Oregon.* S. M. ZELLER.

There occurs in Douglas and Jackson counties, infections of an Exoascus on the wild plum, *Prunus subcordata*, causing "plum pocket." This fungus differs from *E. pruni* and *E. communis*, its nearest affinities, in morphology and specialized host infection. It seemingly does not infect cultivated varieties of Prunus. A complete description of the species will be published soon.

*Relation of rainfall to the late blight of Phoma rot of the sugar-beet.* B. L. RICHARDS.

During the past season of 1921 a definite late blight of the sugar-beet became seriously epidemic throughout the northern part of Utah and southern Idaho. In Cache Valley, with a total acreage of 27,456 acres, 50 per cent of the fields were diseased. The disease in the field varied in severity from a fraction of 1 per cent of the beets destroyed to a total destruction of the crop. Many fields in the district were left unharvested, while others scarcely paid the expense of digging. The loss in the valley alone aggregated approximately two-thirds of a million dollars.

The available evidence indicates that the trouble is possibly identical with Phoma root-rot described by Edson and by various European workers. Data obtained from a study of this and similar but less severe epidemics of the disease in the Logan factory district during 1917 to 1919, show that such epidemics are favored by drought and, locally at least, are closely correlated with abnormally low precipitation during the months of June and July. This lack of rainfall, especially during June, appears definitely to create a critical and dangerous period in the life of the beet which our cultural methods and irrigation practices fail adequately to bridge over.

During certain years, as in 1921, the young beets owing to an abrupt cessation of the spring precipitation in May are forced into an early drought period during which they become so decreased in vitality that they fail to recover and with the advance of the season fall prey to soil organisms responsible for the late blight and root-rot of the crop.

*Respiration of potatoes in relation to the occurrence of blackheart in storage.* J. P. BENNETT AND E. T. BARTHOLOMEW.

Earlier work by Bartholomew and by Stewart and Mix, indicated that blackheart was due to a disturbance of respirational processes dependent on a temperature-time-oxygen relationship. Experiments with measured volumes of tubers and air in sealed chambers at different temperatures decreases with decreasing temperature from 40° C. to 50° C., then increases from 5° C. to 0° C. Decreasing oxygen concentration does not markedly affect the rate of respiration at temperatures below 35° C. until a very low concentration is reached.

The temperature-time-oxygen relation in the production of blackheart appears to be fairly definite. Between 40° C. and 5° C. decreasing temperature increased the period of exposure required to induce blackheart from eight to seventy-seven days; temperature below 5° C. decreased this period. At temperatures below 35° C. blackheart did not occur until practically complete exhaustion of the oxygen in the chamber; with increase of temperature above 30° C. an increasing amount of oxygen remained at the time of appearance of blackheart. Injury leading to the development of blackheart appears to be due to anaerobic processes in the tissues. At temperatures at which exhaustion of oxygen from the chamber precedes the appearance of blackheart, the injury may appear in any part of the tuber; at higher temperatures it usually occurs centrally.

*Potato blackleg with special reference to the etiological agent.* H. M. JENNISON.

Four different species of Bacillus have been described as the etiological agent of the blackleg disease of potatoes. In order to determine the relationships, some 12 strains of the blackleg pathogen, including the 4 "species" above mentioned, were studied comparatively as to morphology, cultural features and physiology. Special attention was given to the comparative study of the gas and acid producing function of the blackleg bacillus and this led to a quantitative study of carbohydrate utilization. An exhaustive examination and study of the literature was also made.

Below is presented a brief summary of some of the conclusions reached.

(1) The strains studied were morphologically similar. That they were specifically identical was abundantly proven by extensive comparative studies of their cultural features and physiology.

(2) The etiological agent of potato blackleg should be referred to as *Bacillus atrosepticus* van Hall.

(3) The following names are to be considered only as synonyms: *Bacillus phytophthorus* Appel., *B. solanisaprus* Harrison, *B. melanogenes* Pethybridge & Murphy.

(4) The Index Number 5312–32120–2110 is presented herewith in lieu of a fuller (revised) description of *Bacillus atrosepticus* van Hall.

(5) This pathogen develops acid and gas in the presence of a number of saccharides. The gas producing function is relatively weak, but this capacity can be built up to a certain extent by constant cultivation in the sugars which it is able to utilize.

(6) Quantitative determinations of carbohydrate utilization show that *Bacillus atrosepticus* cannot hydrolyze potato starch or dextrin.

(7) This organism secretes the enzymes invertase, lactase, maltase, as shown by its action on corresponding substrates.

*Corticium vagum as a factor in potato production.* B. L. RICHARDS.

In a series of pure culture experiments conducted under normal field conditions and in the greenhouse, several strains of *Corticium vagum* were found to produce severe and characteristic cankers on all underground parts of the potato. All strains, though able to infect mature stems either with or without wounding, were found to attack the plant most vigorously in its earlier stages of development. Growing points of the young shoots and of the stolons proved especially susceptible to attack.

In field experiments in which several hundred diseased hills were individually studied and carefully checked against disease-free hills, it was found that the fungus under conditions favorable for its pathogenic activity seriously reduced the numbers and size of the tubers, decreased the number of stems per hill, and greatly weakened the surviving vines. The latter were in general undersize, less vigorous, and died earlier than their disease-free neighbors. A comparison of the final yields from approximately five hundred diseased hills with an equal number of disease-free hills, grown in adjacent rows and under comparable conditions, showed that plants grown in cool soil infected with the Rhizoctonia stage of *Corticium vagum* on the average were reduced in yield to 50 per cent of that obtained from plants freed from the fungus by seed treatment.

Under natural conditions of potato culture, soil temperature proved to be the most important factor in determining loss to the crop.

*Hosts for Puccinia glumarum E. & H. in the United States.* CHAS. W. HUNGERFORD.

*Influence of the meteorological factors on potato disease and production in Colorado.* H. G. MACMILLAN.

Temperature and factors influencing temperature appear to affect the potato yield in Colorado. A critical study of meteorological factors and potato production indicates that temperature for at least six months prior to planting is reflected in the condition of the crop and the yield. Summer temperatures have much less effect. No data are available over many years as to the nature and causes of the prevailing disease, but Fusarium blight probably has been the controlling factor in potato production in Colorado. The high temperature in certain years, showing an increase above the normal during the winter and spring months appears to cause infection of the potato seed pieces or plants by the Fusarium fungus, with a rapid increase in the quantity of disease. An estimate of the winter and spring temperatures, showing whether they are above or below normal would be of great benefit to the farmer in preparing his seed potatoes. In years of high winter and spring temperatures he should plant whole seed. The critical temperature for infection of the potato seedpiece by Fusarium is about 14° C.

*Thielavia basicola on watermelon in Oregon.* M. B. MCKAY.

During the summer of 1916 diseased watermelon plants were received for diagnosis by the experiment station from Grand Island, Yamhill county, Oregon. The older leaves in the center of affected hills were dying prematurely, turning brown, wrinkling and drying up from the center of the hill outward, with the leaves on the outer and younger portions of the runners now about 18 inches long apparently unaffected. The grower suspected a leaf disease but examinations failed to disclose any evidence of such a malady. On the other hand portions of the outer tissues of the main stalk from one to four inches below the ground surface were from slightly to considerably disintegrated, giving them a scurfy appearance. Mounts from this tissue revealed the presence of a considerable number of chlamydospores which on culturing, gave typical chlamydospores and endoconidia of *Thielavia basicola* Zopf. Inoculations of young watermelon seedlings with this fungus in damp chambers have rather vigorous root rot in two week's time. So far as the writer is aware this fungus has not been found previously in Oregon on any plant nor does it appear to have been encountered frequently in the western states. O'Gara reported it as parasitic on watermelons in the Salt Lake Valley, Utah, in 1915.

*Distribution of Tylenchus dipsaci on wild strawberry in Oregon—Preliminary report.* M. B. MCKAY.

During 1921 the leaf and stem-infecting nematode *Tylenchus dipsaci* (Kühn) Bastian was found on wild strawberry plants in the vicinity of Siltcoos Lake in western Lane County. During the two previous seasons this pest had been encountered on cultivated strawberries and also on clover in the same general locality. Considerable interest was therefore attached to the occurrence of the disease on the wild plants, and a cooperative survey was arranged with several agencies participating to determine the approximate distribution of the disease in the coast regions of the state considered most apt to show the malady. To date the disease has been found on wild strawberry plants in four counties, namely Lincoln, Lane, Douglas, and Coos. In Lincoln county the diseased area found was very small, being only a few square yards in extent, and located a few yards from the ocean. In Lane county diseased plants were found scattered over an area about a mile in width and two miles long all within one and one-half miles of the beach. The diseased plants found in Douglas county are really an extension

of the infested area in Lane county and were found only a short distance over the county line. In Coos county diseased plants were found in an area extending back one-half mile from and along the beach for two and one-half miles. In this case the limits of the infestation have not been determined. The survey is being continued.

*Destructive rust (Puccinia subnitens Dietel) on spinach in the northwest.* H. P. BARSS.

On May 4, 1922, liberal samples of spinach leaves were forwarded to the Oregon Experiment Station by J. B. Wiley from the Walla Walla valley. These leaves were abundantly sprinkled with the bright orange cluster cups of a rust. The vegetable growers of the Walla Walla valley have sustained heavy losses on the spinach, both in the early and late crop. No losses from such a disease have been reported to the Oregon Experiment Station in previous years, and growers in the district report no commercial losses from this cause in previous years. Inasmuch as there appears to be no common rust disease accompanying spinach production in most parts of the world it was at once expected that some native rust had attacked the spinach. *Puccinia subnitens* Dietel was suspected and a search for the salt grass or alkali grass *Distichlis spicata* which is the telial host of this rust proved that the alkali grass was one of the three most common grasses growing in the vicinity of the spinach beds.

Specimens of this grass collected by Mr. Wiley close to the spinach beds were completely covered with the telial sori of *Puccinia subnitens*. Cultural experiments at Corvallis under experimental control have demonstrated the ability of the rust sporidia from this material to produce aecial infections on the cultivated spinach.

*Pathology of quaking aspen in Utah in relation to regulation.* E. P. MEINECKE.

Quaking aspen which covers large areas in Utah is at present without value as a timber tree. The growing scarcity of pulpwood points to the possibility of furniture utilization of quaking aspen, under the assumption that the species can be raised to merchantable size before decay destroys too much of the wood produced. The bulk of decay is caused by *Fomes igniarius*. Cull is low in younger age classes and increases with age particularly from the 91–100 age class on. Relative vigor of growth and character of wounding, besides age, influence the cull per cent. About 50 per cent of the wounds become infected. Wounds from fire commonly lead to infection and heavy cull. Elimination of fires and active control of injurious factors through elimination of infected trees are indispensable in the regulation of aspen stands. Since the probable silvicultural rotation of aspen will be shorter than the pathological rotation, it appears possible to raise quaking aspen for pulp in Utah.

*The toxicity of copper sulphate to the spores of Tilletia tritici (Bjerk.) Winter.* FRED N. BRIGGS.

*Stimulation by copper carbonate of wheat seedlings in the greenhouse.* FRED N. BRIGGS.

*Studies on Helminthosporium species found on cultivated barley in California.* G. E. PAXTON.

Studies carried on at Riverside, California, showed the mature perithecial stage of *Helminthosporium gramineum* to be present on two year old barley straw. On March 28, 1922, transfers were made of mature ascospores to cornmeal agar. Typical *Helminthosporium gramineum* conidia were produced. *Hordeum sativum* inoculated with the same gave typical lesions of this species.

Sixteen year old herbarium specimens of cultivated barley affected with *Helminthosporium gramineum*, when placed in a moist chamber, produced, after two weeks, conidiophores and conidia from the dormant mycelium. These conidia germinated readily in water and on potato agar. These conidia measured 100 to 110 by 12 to 15 $\mu$.

The results of tests made with *Helminthosporium sativum* showed the optimum temperature for growth and sporulation to be about 30° C. Corn-meal agar proved to be a more favorable medium for growth than potato agar.

*Helminthosporium sativum* conidia from cultivated barley, *Hordeum sativum*, were grown in pure culture. Transfers from the conidia of this culture were made to squirrel-tail grass, *Hordeum muranium*, producing typical *Helminthosporium sativum* lesions and conidia. The reverse cross from squirrel-tail grass to cultivated barley gave like results.

*A Helminthosporium root rot of wheat in Idaho.* J. M. RAEDER.

A rather serious root rot of wheat was reported from Madison county, Idaho, last year. Investigations of the trouble disclosed the fact that it was confined to the dry land farms. The fields affected were spotted with diseased plants. These spots were irregular in shape and seemed to be confined to low swales or where snow had drifted the previous winter. The plants occurring on these spots were stunted and lighter in color. There was evidence that there had been considerable tillering earlier in the season, most of which had been killed. Counts made in the worst infected spots, showed only one in fifty plants remaining. Heads were formed on the remaining plants, but were very stunted and contained only shrivelled grain.

Closer examination of the affected plants, showed considerable discoloration of the sheath at the base. Occasionally a brownish, triangular shaped spot could be found on the lower first or second internode. Sometimes the lower nodes themselves were discolored.

Isolations were made from infected plants, which consistently gave a species of Helminthosporium. No work has been done to determine the species.

*A Fusarium blight of spinach.* CHAS. W. HUNGERFORD.

A rather serious spinach disease has recently been found in several localities in Idaho. The disease appears when the plants are quite small, causing a stunting of the plants, curling of the leaves and finally the death of the plants. A species of Fusarium has been constantly isolated from the interior of the crowns and roots of diseased plants. The characteristic symptoms of the disease have been produced upon plants grown in sterilized soil to which had been added a pure culture of the organism.

Dr. C. D. Sherbakoff, to whom a culture was sent for identification, has found that the *Fusarium* belongs to the Marteilla-Elegans section and is sufficiently different from all previously described Fusaria to be considered a new species.

*The effect of presprinkling with water upon the efficiency of certain potato seed treatments for the control of Rhizoctonia.* J. M. RAEDER and CHAS. W. HUNGERFORD.

Preliminary laboratory tests conducted at the Idaho Experiment Station have shown that the efficiency of both the corrosive sublimate and the hot formaldehyde treatments for seed potatoes is greatly increased by first sprinkling the potatoes with water and covering for either 24 or 48 hours. Cultures showed that all sclerotia were killed when potatoes were treated as follows: (1) Sprinkled and covered 24 hours then treated with formaldehyde 1 to 120 at 50° C. for 3 minutes. (2) Sprinkled and covered for 24 hours

then treated with formaldehyde 1 to 120 at 55° C. for 1 minute. (3) Sprinkled and covered 48 hours then treated with formaldehyde 1 to 120 at 50° C. for 2 minutes. Cultures were made from similar lots of potatoes which were not presprinkled before treatment with hot formaldehyde showed that control was not absolute.

Sprinkled with water and covering for 24 and 48 hours before treatment increased the efficiency of the mercuric chloride treatments somewhat but in no case was the control absolute. Field tests last year showed increased efficiency of sprinkling previous to treatment with mercuric chloride. Further field tests are being conducted this year.

*White pine blister rust in the Pacific Northwest.* J. S. BOYCE.

White pine blister rust (*Cronartium ribicole* Fischer) was found for the first time in the Pacific Northwest in the fall of 1921. The English black currant (*Ribes nigrum*) was found quite generally infected in the cultivated portion of western British Columbia. A few diseased eastern white and Himalayan pines were discovered in Vancouver.

In Washington, infected black currants were located at Sumas, Mt. Vernon, Everett, and Port Townsend, Two eastern white pines, killed by the parasite, were collected in a nursery at Mt. Vernon.

Scouting in the spring of 1922 has shown infection on native western white pine (*Pinus monticola* Dougl.) in western British Columbia to be general. As yet this spring no infected native pines have been discovered in Washington.

Observations show that the black currant is readily infected with a subsequent heavy spore production. This accords with the behavior of the same host in the east. Furthermore, western white pine seems to be very susceptible.

So far there is no indication of the disease in the commercial white pine region of Idaho or western British Columbia or in the sugar pine stands of southern Oregon and British Columbia. Every effort must be made to keep these regions free from infection.

*Symposium*: The relation of temperature and other factors to the etiology of plant diseases.

Discussion by Dr. C. W. Hungerford, University of Idaho, and Dr. H. S. Fawcett, Citrus Experiment Station. This symposium was held jointly with the Plant Physiologists.

*The August number of Phytopathology was issued September 11, 1922.*